DISSERTATION ON STUDY OF CORRELATION OF ANTHROPOMETRIC MEASUREMENTS WITH DIABETIC RETINOPATHY

Submitted to
The Tamil Nadu Dr. M.G.R. Medical University
In partial fulfillment of regulations for the award of the degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE

KILPAUK MEDICAL COLLEGE

CHENNAI – 10

THE TAMIL NADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2013

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON CORRELATION OF ANTHROPOMETRIC MEASUREMENTS WITH DIABETIC RETINOPATHY "** is a bonafide work done by Dr.**K.DEVI,** Post Graduate student, Department of General Medicine, Kilpauk Medical College, Chennai - 10, under our guidance and supervision in partial fulfillment of the Rules and Regulations of **The Tamilnadu Dr.M.G.R.Medical University** for the award of **M.D.Degree Branch I, General Medicine** during the Academic period from May 2010 to April 2013.

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DECLARATION

I solemnly declare that the dissertation entitled "**A STUDY ON CORRELATION OF ANTHROPOMETRIC MEASUREMENTS WITH DIADETIC RETINOPATHY"** was done by me at Kilpauk Medical College, Chennai under the able guidance and supervision of **Prof. Dr.K.T.JAYAKUMAR, M.D.,** Professor, Department of General Medicine, Government Royapettah Hospital, Chennai.

This dissertation is to submitted to **The Tamilnadu Dr. M.G.R. Medical University, Chennai** towards the partial fulfillment of requirements for the award of the degree of **M.D. Branch -I General Medicine**.

Dr.K.DEVI

Place:
Date:

ACKNOWLEDGEMENT

I sincerely thank **Prof.Dr.P.Ramakrishnan, M.D., D.L.O.,** Dean, Kilpauk Medical College, Chennai for permitting me to utilize the facilities needed for this dissertation work.

I would like to express my special thanks to beloved **Prof.Dr.N.Gunasekaran, M.D., D.T.C.D.,** Director and Superintendent, Institute of Non Communicable Diseases, Government Royapettah Hospital, Professor and Head of the Department of General Medicine, Kilpauk Medical College and Hospital, Chennai for permitting me to carry out this study and for his constant encouragement and guidance.

I also express my sincere gratitude to **Prof.Dr.K.T.Jayakumar, M.D.,** my Unit Chief and Professor of Medicine for his guidance and constant support during the entire period of my study.

I am immensely thankful to **Prof.Dr.R.Sabaratnavel, M.D., Prof.Dr.S.Mayilvahanan, M.D.** for their help and guidance rendered during the entire period of my work.

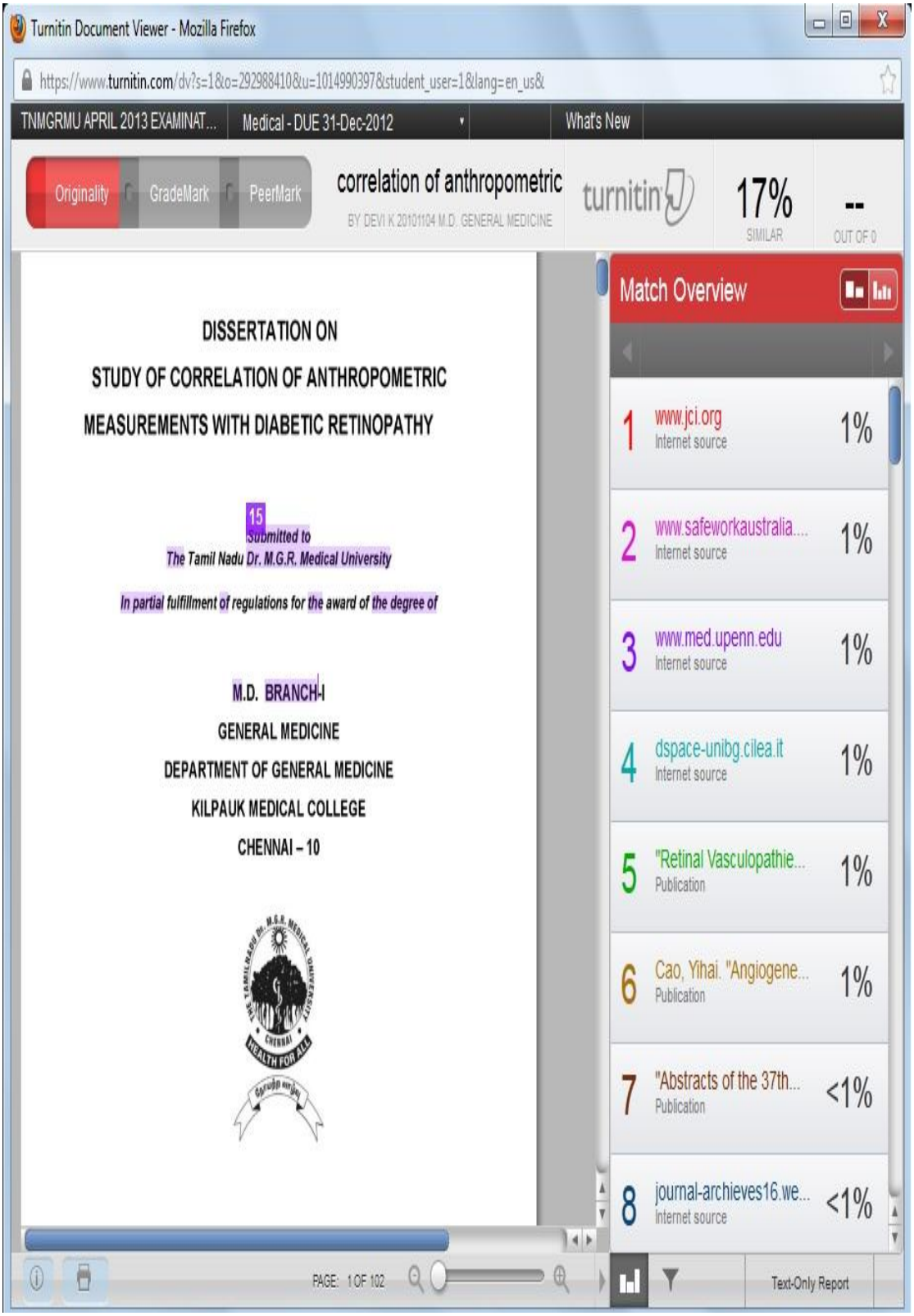
I wish to thank **Dr.K.Manickam**, **M.D.,** Medical Registrar**, Dr Shaik Sulaiman Meeran M.D, Dr.T.Ramesh Kumar M.D, Dr.K.E.Govindarajullu M.D,** Assistant Professors, Department of Medicine, Government Royapettah Hospital for their valuable suggestions and support throughout this work.

I also wish to thank **Prof. Dr R. Mani M.S.,** and Assistant professors **Dr.R.Saravanan** and **Dr.Aruna** of department of ophthalmlology for their constant guidance and support.

I also extend my thanks to the Statistician and all the laboratory technicians for their valuable support throughout my dissertation work.

I also thank my parents **Mr.P.Krishnan**, and **Mrs.P.Manimegalai** and my husband **Dr.K.Senthil Kumar**, siblings, colleagues, friends and staff of our hospital for their support of this work.

Last but not the least, with sincere gratitude, I thank all the patients who contributed so much to this study.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CONTENTS

INTRODUCTION

INTRODUCTION

"Diabetes is a dreadful distress and burden, where flesh and limbs melts into urine, patients don't stop drinking water and the flow is continuous without interruption like opening of aqueduct." Life is disagreeable and full of discomfort, thirst is impossible to satisfy, an insatiable appetite, passing litres of urine, not appropriate to the intake of fluids. If they refrain from drinking, their mouth seem dry and body gets roasted and their viscera seems to wither or parch with intense heat and excoriate as pointed in second century A. D by Aretaeus of Cappadocia.

Diabetes mellitus is a disease of ancient times which is a complex metabolic disorder characterised by chronic hyperglycemia contributed by the dysregulation of carbohydrate, fat and protein metabolism due to decreased secretion of insulin, defect in the utilization of glucose, and increased production of glucose.[1] World Health Organisation classified diabetes mellitus according to the etiology into type 1, type 2 , gestational diabetes and other types.[2]

Diabetes mellitus imposes an increased global burden contributing to both micro vascular complications like retinopathy,

nephropathy, and macro vascular complication like stroke and coronary artery disease.It is the leading cause of vision loss[3], lower limb amputation[4], renal failure. Globally it is the fifth major cause of death.

In 2010, 285 million people were suffering from this disease and hence no doubt it is the most common non communicable disease globally due to changing cultural habits and rapid westernisation. Prevalence of diabetes in India is 62.4 million and hence this study was done to analyse causal association of obesity with diabetic complication namely diabetic retinopathy.

The pathogenesis of micro vascular complication is through chronic intracellular hyperglycemia and the tissues are affected by various mechanisms like more influx of glucose through the polyol pathway, increased production of advanced glycated end products and raised formation of free radicals in mitochondria. One such important micro vascular complication of diabetes is retinopathy.

Diabetic retinopathy causes frightening vision loss in working age. Though, cataract and refractive error were the leading cause of impaired vision in Asia, as the proportion of diabetes mellitus multiplies here in Asia, diabetic retinopathy has to be thoroughly screened for, as per the saying "prevention is better than cure."Diabetic patient have to be

screened periodically to avoid the untoward consequence of vision loss due to diabetic retinopathy.

World Health Organization has published a statistical report that diabetic population in Asia especially in India and China is 52.4 million. This number is forecasted to multiply to 121.8 million by 2030,[5] due to the lack of knowledge and information regarding the modifiable risk factors. By 2030, the number of senior citizens is expected to climb by 168 percentage in Asia [5] and due to the increasing prevalence of diabetes mellitus in the younger generation due to changing cultural habits, westernization allows the time interval for the diabetic patients to develop complications.

Thus diabetes imposes an economic burden on the national health policies, so early identification and modification of the risk factors would definitely help to reduce financial burden of the individual as well the country. This study was conducted with the aim to find the correlation of anthropometric measurements (height, weight, BMI, waist circumference, hip circumference, waist hip ratio, neck circumference) and serum lipids with the severity of diabetic retinopathy.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The name “diabetes” is taken from the Greek word for a syphon; the sweet taste of urine of the diabetes was recognized in the beginning of the first millennium, and the adjective “ mellitus ” meaning (honeyed) was given by Rollo in the 18th century.

EPIDEMIOLOGY OF DIABETES MELLITUS:

Current estimates suggest that two - thirds of those affected by diabetes live in lower and middle economic group countries.The pandemic growth of diabetes is due to transitional change in demographic, nutritional socioeconomic, lifestyle pattern. The International Diabetes Federation quotes that there are 285 million people with diabetes globally in 2010, and projects that the absolute number will increase beyond 400 million in 2025. The percentage of diabetes will increase by 170% in the low and the middle income countries, compared to a 41% in developed countries.

The prevalence of DM has increased from 30 million to 285 million, since 15 years from 1995. Even though type 1 & type 2 diabetes increases widely across the globe, the incidence of type 2 diabetes mellitus has increased dramatically due to increasing over weight and

obesity, decreased physical activities, rapid urbanization and increase in number of older generation. Newly detected diabetics in 2010 were 1.6 million. In 2010, the incidence of DM in United States of the America was estimated to be 26.9% in the age group greater than 65 years. The prevalence of diabetes was almost equal in both the sexes. It is estimated that in 2030 there will be more number of diabetics under the category of 45 to 60 years. Much geographical difference is seen higher incidence of type 1 diabetes in Scandinavian countries and lower incidence seen in the Pacific zone.

DIAGNOSIS OF DIABETES [1]

- "Symptoms of diabetes and random blood glucose concentration -11.1 mmol/L or 200 mg/dL[a] *or*
- Fasting plasma glucose -7.0 mmol/L or 126 mg/dL[b] *or*
- HbA1C > 6.5%[c] *or*
- Two-hour plasma glucose -11.1 mmol/L or 200 mg/dL during an oral glucose tolerance test.[d]

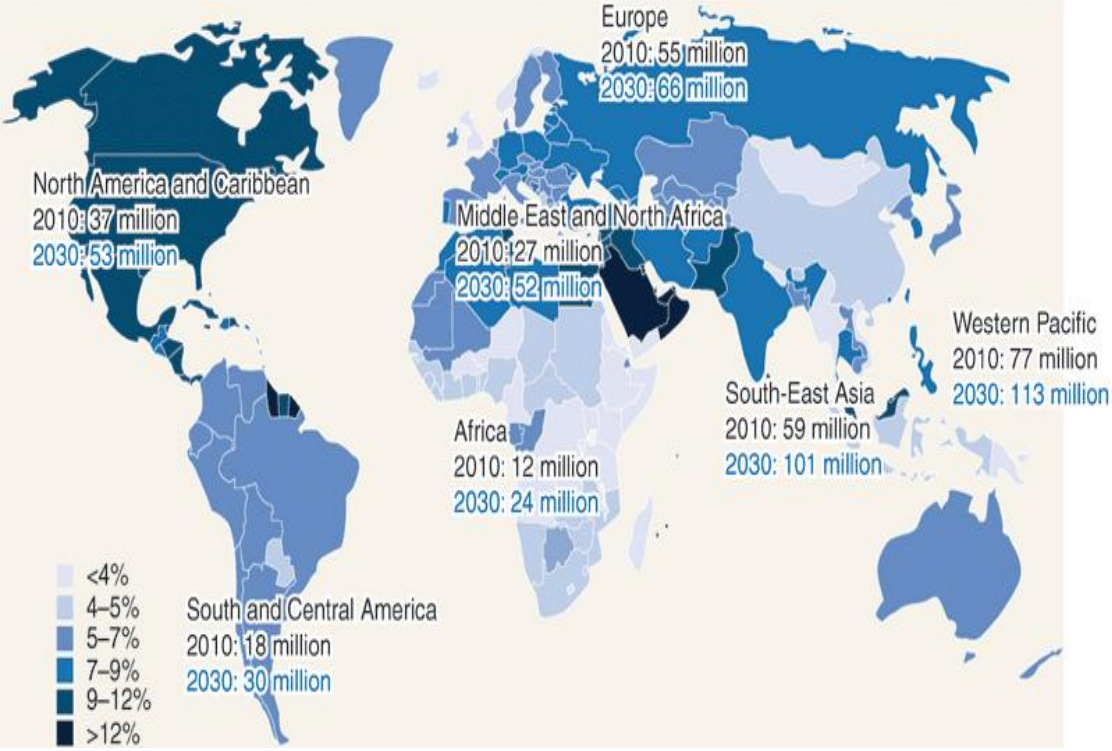
[a] Random - without regard to time since the last meal.

[b] Fasting - no caloric intake for at least 8 h.

[c] The test should be done in lab which is certified according to A1C standards of the DCCT.

[d] The test should be done using a glucose load containing 75 g anhydrous glucose, dissolved in water.

GLOBAL PREVALENCE OF DIABETES MELLITUS

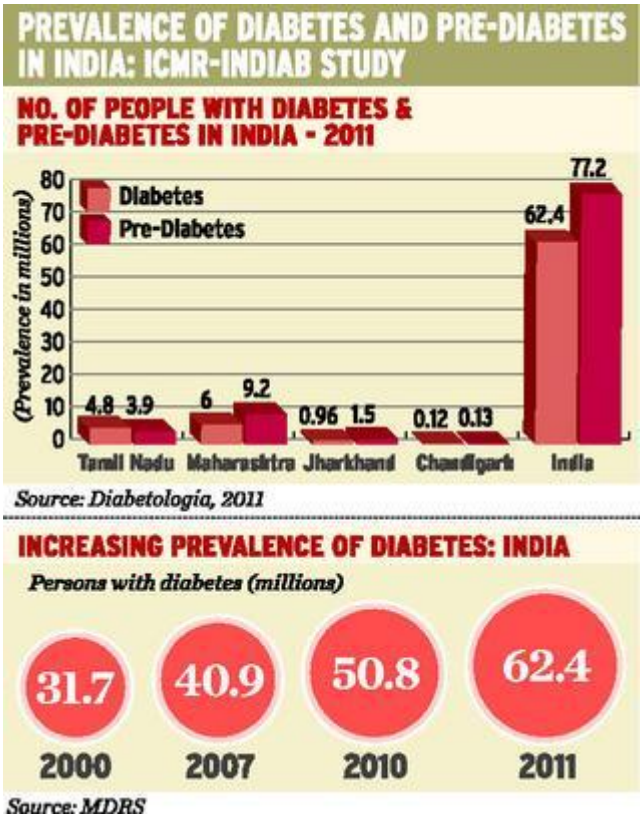


PREVALENCE OF DIABETES IN INDIA

According to Indian Council of Medical Research – India Diabetes (ICMR-INDIAB)(2008-2010)

DIABETES	62.4 MILLION
PRE DIABETES	77.4 MILLION

Phase one project of this study included 3 States and 1 Union Territory, representing nearly 18.1 per cent of the nation's population.



PREVALENCE OF DIABETES IN INDIA

MODIFIABLE BEHAVIOURAL RISK FACTOR

Reduced physical activity

"Insufficient physical activity is defined as less than five times, thirty minutes of physical activity in a week, or less than 3 times, twenty minutes of vigorous activity in a week, or equivalent".Reduced physical activity which is the 4th major risk factor responsible for death. [7] 3.2million deaths each year were associated with insufficient physical

activity.[7] Decreased physical activity has twenty to thirty % increased risk of mortality compared to those who carry out at least thirty minutes of intense physical exercises in most days of the week. [8]

PREVALENCE OF PHYSICAL INACTIVITY GLOBALLY

Gender	Percentage
ADULT MALE	28
ADULT FEMALE	34

Changing Unhealthy dietary habit

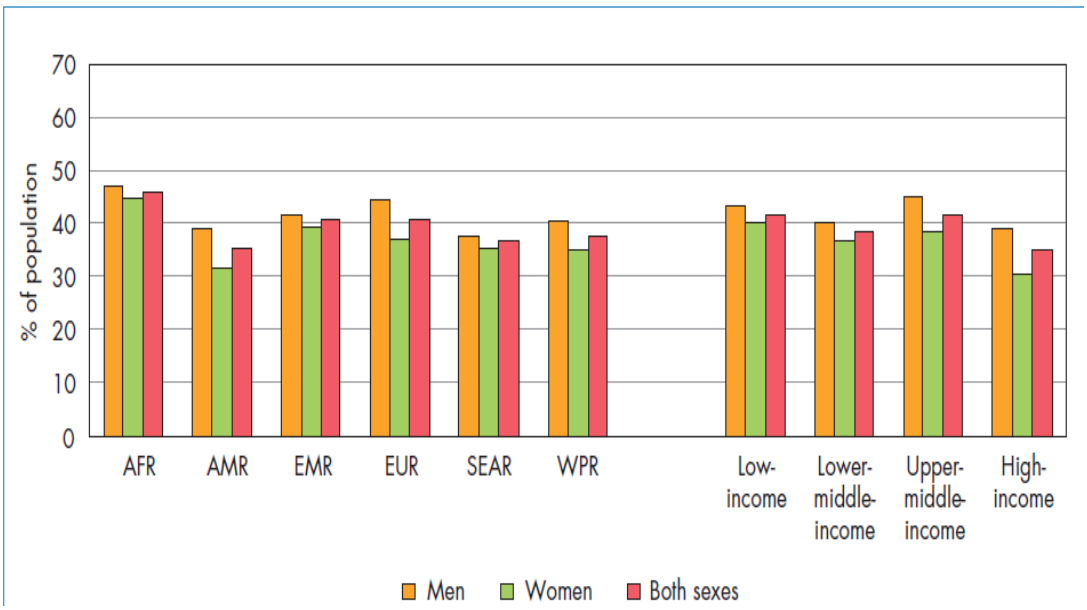
1.7 million deaths globally are due to low fruit, vegetable consumption.[9] Adequate intake of fruit and vegetables decreases the risk for cardiovascular diseases, stomach cancer and colorectal cancer.[10,11]WHO recommends salt intake of less than 5 grams /person /day for prevention of cardiovascular disease. [12] Risk of type 2 diabetes is directly related with intake of saturated fat and trans-fat and inversely related with polyunsaturated fat. [13]

METABOLIC AND PHYSIOLOGICAL RISK FACTORS

Raised blood pressure

"Increased blood pressure is defined as systolic blood pressure of greater or equal to 140 mmHg and or diastolic blood pressure of greater or equal to 90 mmHg"**.** Globally high blood pressure leads to 7.5 million deaths. Raised blood pressure is a risk factor for cerebrovascular accident and ischemic heart disease. [15] The risk of IHD increases twice for every raise of 20/10 mmHg of BP, starting as low as 115/75 mm hg. Across the world the prevalence of increased blood pressure in adults greater than or equal to 25 years was about 40% in 2008. The number of people with hypertension increased to 1 billion in 2008.[16] Men had a higher prevalence than women 39% and 32% respectively.

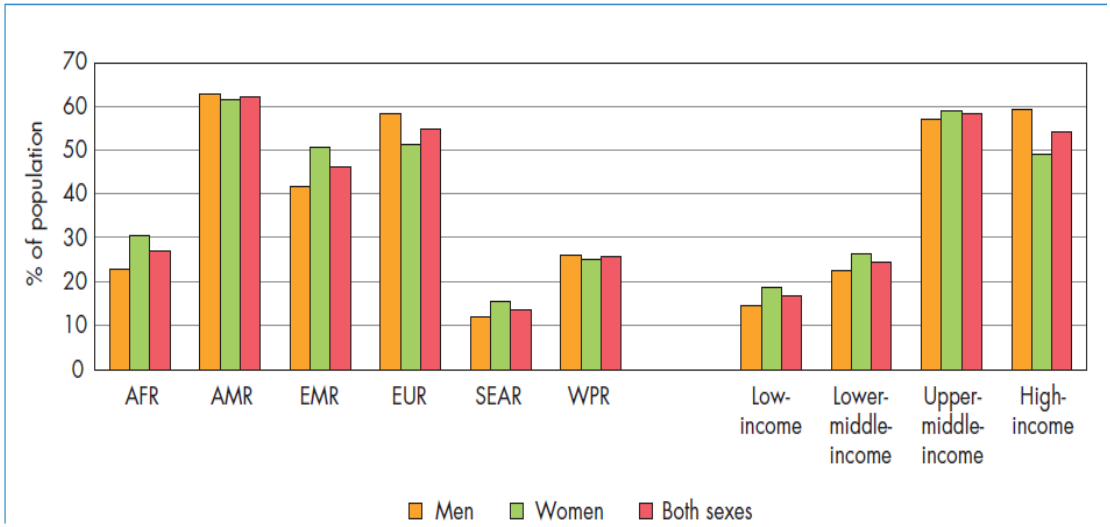
Figure 14. Age-standardized prevalence of raised blood pressure in adults aged 25+ years, by WHO Region and World Bank income group, comparable estimates, 2008



Overweight and obesity

Globally, every year about 2.8 million people die due to obesity and its complications. This leads to worse metabolic effects like increased BP, cholesterol, triglyceride and metabolic syndrome. Risks of IHD, cerebro vascular accidents and diabetes mellitus increases with increase in BMI. Central adiposity is an important feature of the metabolic syndrome, which reflects that it has got close relationship between waist circumference and adiposity.

Figure 15. Age-standardized prevalence of overweight in adults aged 20+ years, by WHO Region and World Bank income group, comparable estimates, 2008



High cholesterol

High cholesterol levels increases the risk of heart disease and stroke.[17] World wide, one third of IHD is associated with raised cholesterol. It accounts for 2.6 million deaths.

GENDER	RAISED CHOLESTEROL (%)
ADULT MALE	37
ADULT FEMALE	40

METABOLIC SYNDROME

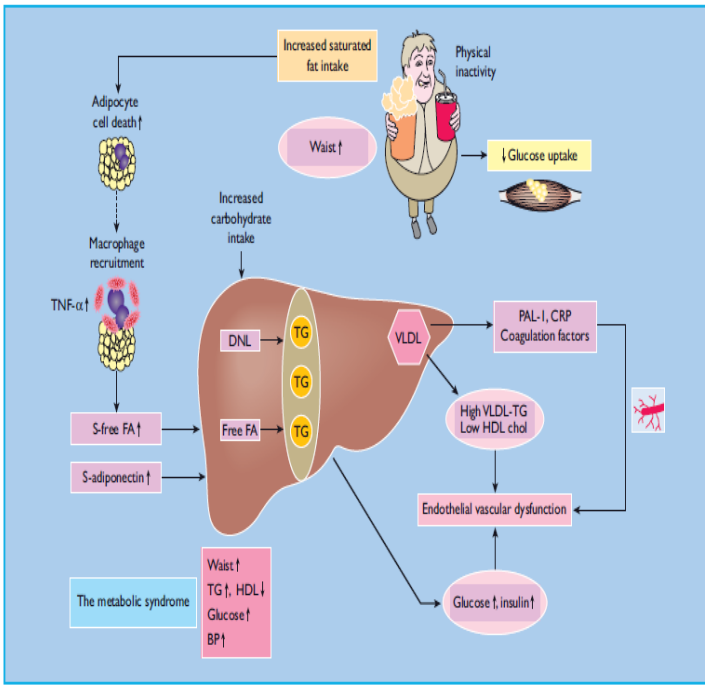
Also called as syndrome X or insulin resistance syndrome. It is a group of metabolic abnormalities that serves as a risk factor for development of cardiovascular morbidities and mortalities and diabetes mellitus (DM). Criteria for metabolic syndrome was given by World Health Organization. The major features of the metabolic syndrome include

- central obesity,
- hypertriglyceridemia,
- low high-density lipoprotein (HDL)
- cholesterol,
- hyperglycemia,
- and hypertension.

PATHOGENESIS OF METABOLIC SYNDROME

Expanded adipose tissue mass releases free fatty acids (FFAs) in abundance. Increase in FFAs results in an abundant production of glucose and triglycerides and secretion of Very Low Density Lipoproteins (VLDLs) in liver, reductions in High-Density Lipoprotein (HDL) and an increased density of Low-Density Lipoproteins (LDLs). Insulin sensitivity is reduced in muscle by inhibiting insulin-mediated uptake of glucose, as a result glucose conversion to glycogen is reduced and increased accumulation of lipid in triglyceride (TG). Hyperinsulinemia occurs due to circulating high glucose, FFA and increase in pancreatic insulin secretion.

Hyperinsulinemia will result in increased reabsorption of sodium in the proximal convoluted tubules of nephron which stimulates the activity of sympathetic nervous system results in hypertension, and in turn resulting in increased levels of circulating FFAs that superimposes on the pro inflammatory state and contributes to the insulin resistance through increased production of interleukin 6 and tumor necrosis factor by adipocytes and monocytes. Prothrombotic state occurs due to increased hepatic production of fibrinogen and production of plasminogen activator inhibitor 1 (PAI-1) by the adipose tissue. The metabolic syndrome is associated with decreased production of the anti-inflammatory and insulin-sensitizing cytokine like adiponectin



CAUSES AND CONSEQUENCES OF METABOLIC SYNDROME

National Cholesterol Education Program criteria for metabolic syndrome

Three or more of the following

- Central obesity: Waist circumference greater than 102 cm in males and 88 cm in females.

- Hypertriglyceridemia: Triglycerides greater than or equal to 150 mg/dL or specific medication.

- Low HDL cholesterol less than 40 mg/dL and 50 mg/dL, respectively in male and female or specific medication.

- Hypertension: Blood pressure greater than or equal to 130 mm Hg of systolic and 85 mm Hg of diastolic or specific medication.

- Fasting plasma glucose greater than or equal to 100 mg/Dl or specific medication or previously diagnosed Type 2 diabetes.The risk factors are age, overweight, obesity, sedentary life style, coronary heart disease, diabetes, dyslipemia, and hypertension.

INTERNATIONAL DIABETES FOUNDATION [IDF] CRITERIA – SOUTH ASIANS FOR METABOLIC SYNDROME:

Two or more of the following

- Waist circumferences in adult males is ≥ 90 cm and ≥ 80 cm in females . Fasting triglycerides greater than 150 mg/dL or specific medication.

- HDL cholesterol less than 40 mg/dL and 50 mg/dL for men and women, respectively, or specific medication.

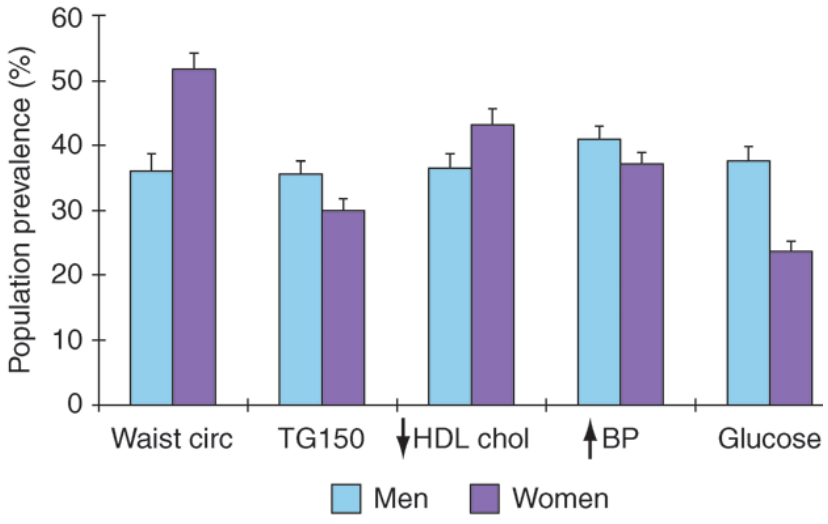
- Blood pressure greater than 130 mm Hg systolic or greater than 85 mm Hg diastolic or previous diagnosis or specific medication.

- Fasting plasma glucose greater than 100 mg/dL or previously diagnosed Type 2 diabetes.

PREVALANCE OF CENTRAL OBESITY IS COMMON IN ASIANS

Compared with the Western population,Asian Indians have higher visceral fat higher upper body adiposity for a given BMI. Indians were found to have more upper body fat, which is valued by the waist-to-hip ratio (WHR) or waist circumference (WC), although they have lean body mass.[19,20,2122] So, normal cut off value for each population varies, so this criteria does not hold good for all races. In northern region of India, the normal BMI was found to be 22 kg/m^2 by Dudeja et al. The WHO states that Asian Indian have lowers limits of BMI. Urban Indians have healthy BMI of 23 kg/m^2, and the cut off values for waist circumference were 85 cm for men and 80 cm for women and for waist hip ratio were 0.89 and 0.81 for men and women. Hence, waist circumference is an useful index for upper-body adiposity.

POPULATION PREVALENCE OF METABOLIC SYNDROME AND ITS COMPONENTS[18]



Obesity and over weight

Abnormal and enormous fat accumulation may impair the system of health.

Body mass index (BMI) is based on weight-for-height which is commonly used for classification of overweight and obesity in adults.

BMI=weight in kilograms /square of his height in meters (kg/m^2)

Normal body mass index -18.5 to 24.9 kg/m^2.

In adults, when BMI -25 to 29.9 kg/m^2 is termed as overweight.

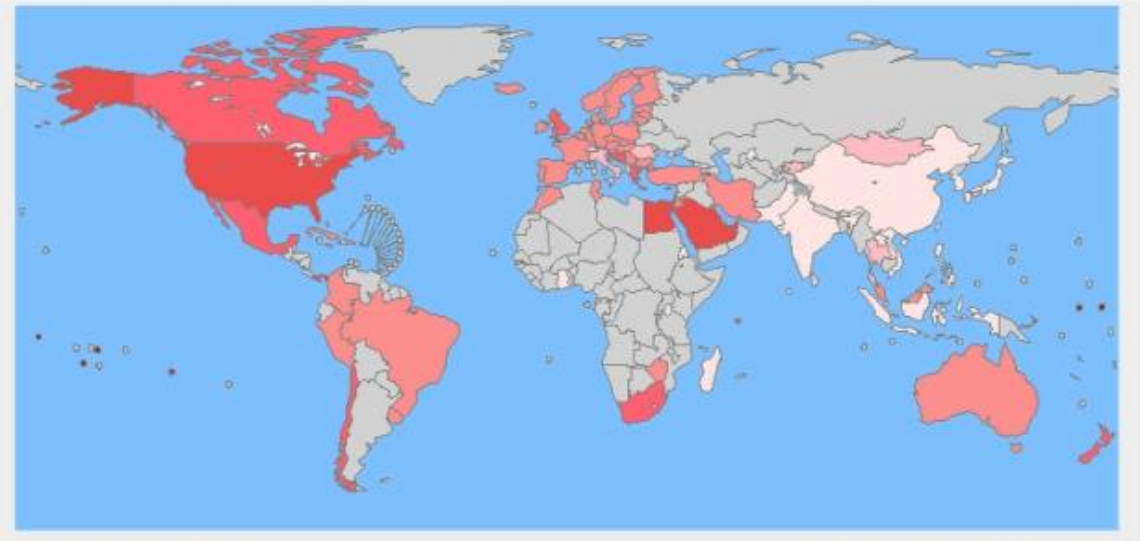
In adults, when BMI is $\geq$ 30 kg/m^2 is termed as obese.

PREVALENCE OF OBESITY AND OVER WEIGHT

In 2010 according to IASO/IOTF analysis, 1.0 billion adults were overweight, and 475 million were obese. In Asians, for obesity whose BMI $\geq$ 28 kg/m^2, then globally, number of adults who are considered obese will be > 600 million!

According to IASO/IOTF, 200 million school going children belong to either overweight or obese category, among them 40-50 million are obese. 300 million women were obese and 200 million men were

obese. In 2010, overweight children under 5 years of age were 40 million. 2.8 million adults die due to obesity/year. Out of which 44% have the DM, 23% have IHD and 7% and 41% of cancer.



WORLD HEALTH ORGANISATION -2011, GLOBAL PREVALENCE OF OBESITY.

OBESITY 40% (PACIFIC ISLANDS)

OBESITY 30-40%

OBESITY 20-30%

OBESITY 10-20%

< 10%

Globally, overweight and obesity causes more mortalities than underweight.

CAUSE OF OBESITY.

Worldwide, there is high consumption of energy-rich foods which are rich in salt, sugar and fat and reduced mineral, vitamins. Reduced exercises due to the highly sedentary nature, urbanization of rural areas sector, agriculture sector are the major cause of obesity.

NUTRITION – DOUBLE EDGED WEAPON

Low and middle economic group countries are affected much with overweight and obesity. Both under-nutrition and obesity exist together within the same country, community and the household.

In developing countries, children are more vulnerable to improper and poor intake of nutrition in pre-natal period, infancy and young childhood. Now a days, children are exposed to high-fatty food, high-sugar, energy-dense, micronutrient-poor foods, which are low in cost.These type of diet patterns along with reduced physical activity, may result in rapid increase in obesity in children.

GENETIC CHANGES IN OBESITY AND DIABETES

Mutation in leptin – melano cortin signaling pathway, results in obesity. 2 -4 % of monogenetic obesity in children is due to mutations in melanocortin - 4 – receptor gene. Among the 20 genetic variants ,variants found near the *FTO* and the *MC4R* gene seem to affect the body weight

in large proportion, resulting in extra 3 kilos compared to other genetic variant[23], which encodes 2-oxoglutarate-dependent nucleic acid demethylase which is expressed in the brain and in the arcuate nucleus of the hypothalamus [24]. Current assumption is that, there is a genetic failure of the pancreas to compensate for insulin resistance, characteristically resulting in obesity, and hence development of Type 2 Diabetes[25].

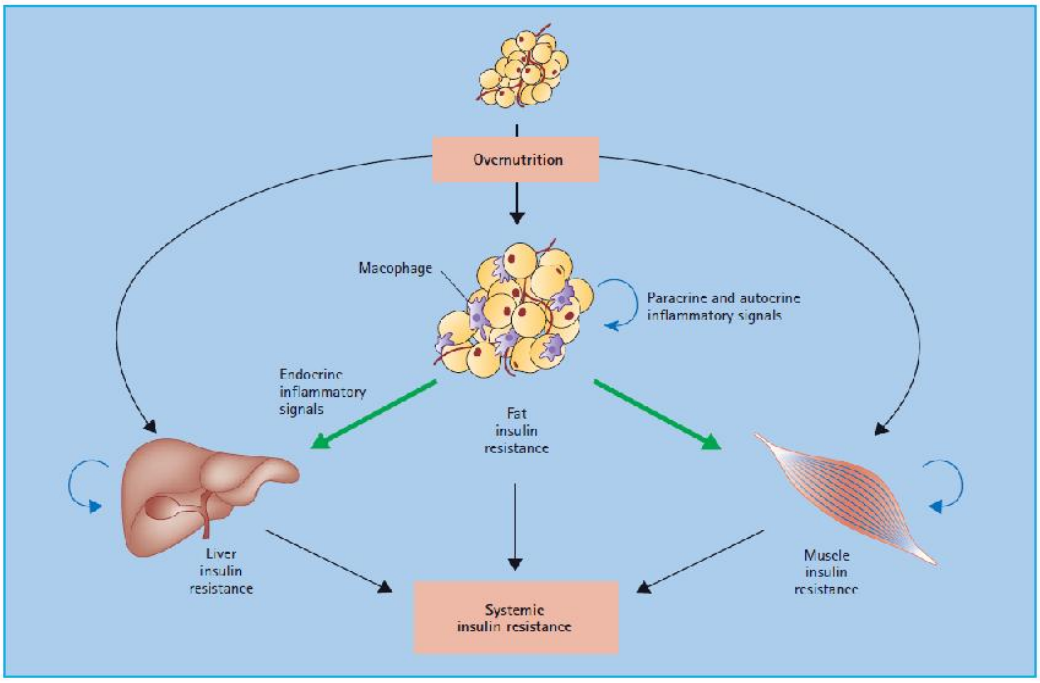
Chronic over nutrition and hyper glycemia during pregnancy results in fetal hyper insulinemia, hyper leptinemia and hyper cortisolemia. These changes leads to mal programming and hence mal functioning of hypothalamic centers which controls the homeostasis of energy and metabolism, which increases the lifetime risk for obesity and T2DM[26]. Gut hormones, signals the gastro intestinal nervous system and induces the complex integration of the central nervous system signalling namely the anorexigenic leptin – melanocortin link and the orexigenic NPY – AgRP pathway, according to the dietary intake of the individual.

LINKS BETWEEN OBESITY AND TYPE 2 DIABETES

INSULIN RESISTANCE AND OBESITY:

The initial hypothesis which relates obesity and T2DM is "glucose – fatty acid cycle" where exists a competition between oxidation of glucose and fatty acid in the muscle[27]. Increased amount of non -

esterified fatty acids from increasing adipose tissue competes with the utilisation of glucose especially in muscle, where glucose is oxidised. Inhibition of glycolytic enzymes like pyruvate dehydrogenase, hexokinase, phosphofructokinase take place. Recent studies suggest that there are more intra myocellular lipid accumulation which also increases the insulin – resistant state. Hence, intra muscular accumulation of neutral fatty acids, and its metabolites such as ceramide, diacylglycerol and fatty acyl coenzyme A (CoA) take place disturbing the insulin action by an activation of a serine –threonine kinase cascade resulting in phosphorylation of Insulin Receptor Substrate-1 (IRS - 1) and IRS – 2. [28]



OVER NUTRITION AND SYSTEMIC INSULIN RESISTANCE

LIPIDS AND BETA - CELL FUNCTION:

Chronic exposure of fatty acids results in impaired insulin secretion and decrease in insulin biosynthesis[29]. Increased expression of uncoupling protein (UCP - 2) results in impaired insulin secretion.

Insulin resistant offspring of parents with T2DM have features of impaired mitochondrial function [30]. Obese individuals have very smaller mitochondria who have reduced bioenergetic capacity when compared with lean controls. [31]

SECRETORY FUNCTION OF ADIPOSE TISSUE .

TNF - α is a cytokine, expressed in adipose tissue [32] leading to suppression of GLUT 4, lipoprotein lipase expression, increase in lipolysis [33], activation of NF - κB leading to increased pro-inflammatory cytokines like Interleukin 6 (IL - 6), IL-8 and monocyte chemotactic protein 1 (MCP). It reduces the expression of adiponectin, which exerts direct antidiabetic and the anti-atherosclerotic actions. It also stimulates the phosphorylation of IRS - 1 at the serine residue 307 there by inhibiting the transduction of the insulin signal [34]. Prodiabetic action include activation of plasminogen activator inhibitor 1 (PAI - 1), lipid metabolites such as ceramide.

Retinol - binding protein 4 (RBP - 4) leads to insulin resistance via reduced PI 3 and anti - inflammatory properties like adiponectin, IL - 1receptor antagonist and IL – 10, adiponectin. Adiponectin levels are inversely associated with BMI and that low level results in the development of T2DM [35,36]. It has an antidiabetic and anti – atherosclerotic activity which increases the oxidation of the fatty acid through an AMP - activated protein kinase pathway.

INFLAMMATORY RESPONSE IN ADIPOSE TISSUE

Inflammatory activity in adipose tissue is through the activation of the c - Jun N – terminal kinase (JNK) and IKK β – NF - κ B, which further stimulates cytokines such as TNF - α and IL – 6.. In obesity, JNK activity is elevated in liver and muscle as in the adipose tissue. IKK β stimulates the production of many pro inflammatory mediators including TNF - α and IL-6 the activation of NF - κ B pathways [37]. The functional capacity of the endoplasmic reticulum is overloaded in obesity and leads to activation of the inflammatory signalling pathways.

OXIDATIVE STRESS

Increased production of reactive oxygen species (ROS) occurs in adipose tissue with increased expression of NADPH oxidase and reduced expression of antioxidative enzymes.

HYPOXIA IN ADIPOSE TISSUE

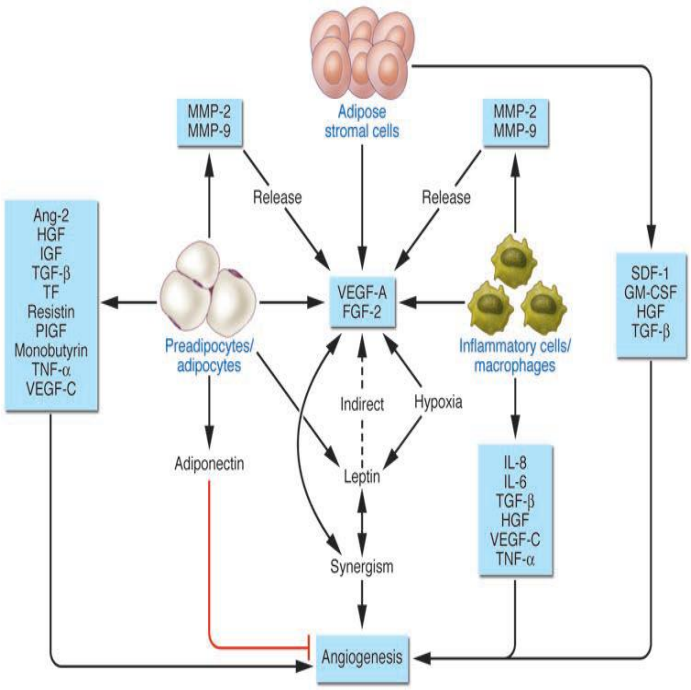
Hypoxic response genes are hypoxia-inducible factor 1α (HIF-1α). Vascular Endothelial Growth Factor (VEGF) and heme oxygenase. It leads to reduced mitochondrial respiration and increase in lactate production. It also reduces the expression of adiponectin.

BODY FAT DISTRIBUTION IN OBESITY

Abdominal type of body fat distribution leads to increased risk of diabetes and cardiovascular complications. Intra - abdominal fat is lipolytically more active than adipocytes in the sub cutaneous tissue and with increased accumulation of lymphocytes and macrophages, indicates the proinflammatory activity. Visceral adipose tissue has greater blood vessel and nerve density with higher metabolic activity which drains into the portal vein. Liver is directly exposed to fatty acids and proteins which stimulates insulin resistance in the liver. The largest endocrine gland is adipose tissue as it produces free fatty acids, growth factors, hormones, and cytokines such as VEGF, HGF, IGF-1, leptin, angiogenin, IL-6, TNF-alpha adiponectin, resistin and angiopoietins.

ADIPOGENESIS AND ANGIOGENESIS

Brown adipose tissue has a very high energy expenditure, but it remains functionally silent in obesity. Its thermogenic activity is due to high perfusion of blood in order to provide O_2 and substrates and to dissipate energy in the form of heat. Hence, angiogenesis is very much necessary for hyperplasia of brown adipose tissue, which depends on the quick activation of mitosis of precursor cells of brown adipose tissue to generate new capillaries. White adipose tissue can be transformed into brown adipose tissue on chronic exposure to cold. Regression of certain capillary networks leads to transformation of brown adipose tissue into white adipose tissue.



ADIPOSE TISSUE MODULATING ANGIOGENESIS

INTERACTION BETWEEN ADIPOCYTES AND ENDOTHELIAL CELLS

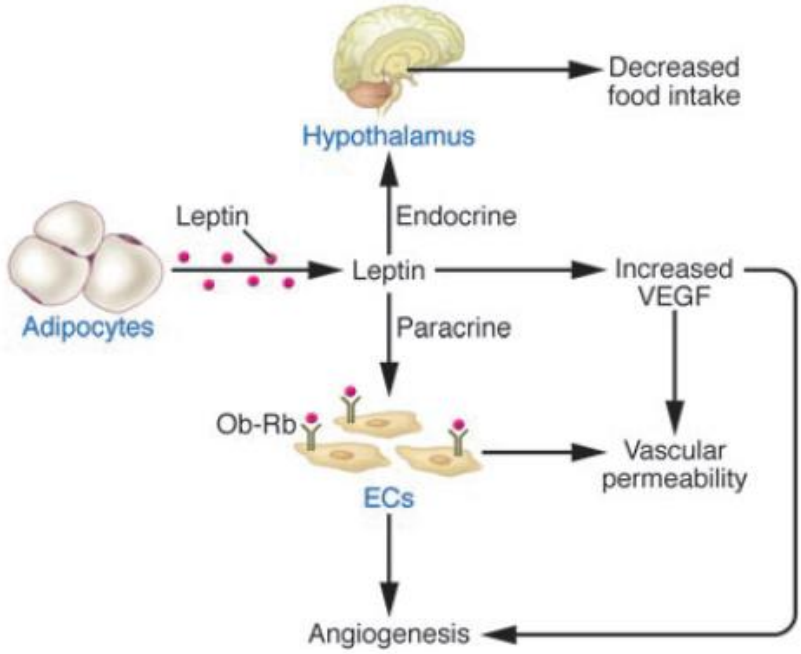
Endothelial cells of the capillaries interact with the fat tissue by means of paracrine signals through ECM, and direct cell-cell communication [39,40,41]. Preadipocytes of human and endothelial cells of the capillaries express plasminogen activator inhibitor 1 and αvβ3 integrin, which helps in preadipocyte migration towards the developing capillaries to enable the coordination of the development of both preadipocytes and the capillary endothelial tissues at the same place.[47]Adipose tissue produces Matrix MetalloProteinases like MMP-2 and-9,and affects the development of microvessel and preadipocyte differentiation by changing the extra cellular matrix [44]. It is able to generate the matrix bound vascular endothelial growth factor and there by stimulates proliferation of blood vessels [45]. Endogenous matrix metallo proteinases stimulate and maintain proliferation of blood vessels[46,47].

High-calorie diet induced obesity is observed in matrix metallo proteinase-3–deficiency. The density of blood vessels in adipose tissue was found to be increased in those mice which were devoid of this particular gene, implying that matrix metallo proteinase -3 alters proliferation of blood vessels in adipose tissue [48]

Tissue inhibitor metallo proteinase and matrix metalloproteins has a major role in angiogenesis. In expanding adipose tissue, hypoxia is found to be one of the major determinant factor for growth of vessels. As a result of hypoxia,adipose tissues produces hypoxia inducible factor 1a– angiogenic factors such as VEGF, leptin, TNF-α, and PAI-1, which helps in the maintenance and regulation of angiogenesis and adipogenesis. Growing adipocytes produce numerous factors which alters the angiogenesis which include leptin, VEGF, VEGF-C, HGF, IGF FGF-2, TGF-β, TNF-α, placental growth factor (PlGF, resistin, heparin-binding epidermal growth factor, neuropeptide .Macrophages which are activated produce more potent angiogenic factors like as TNF-α, VEGF, IL-1b, IL-6, and IL-8 and FGF.

LEPTIN

It is hormone derived from adipose tissues. It governs intake of food and homeostasis of energy. Any impairment in the functioning of leptin results in severe obesity, infertility and diabetes. It is an important factor that influences the proliferation of blood vessels. Endothelial cells express leptin receptor (OB-Rb) – which has helped in discovery of angiogenic activity of leptin[50].In endothelial cells, leptin interacts with its OB-Rb receptor which leadto stimulation of the Stat3 pathway and enhances the activity of its binding .



LEPTIN REGULATING ANGIOGENESIS

Apart from the angiogenic activity of leptin, it also stimulates the expression of VEGF mRNA through the regulation of the Jak/Stat3 signaling pathway. Leptin helps in induction of MMP-2 and MMP-9 activity, which indirectly helps in angiogenesis. During the hypoxic conditions, the ratios of VEGF and leptin are elevated. Thus it has an important role in the development of vasculature and its remodelling by altering the production levels of angiogenic factors.

Leptin, effectively induces the activity of endothelial NOS thereby increasing the vasodilation and also blood supply in the fatty tissue [51,52]. Thus the rapidly developing adipose tissue has many capillaries that are

fenestrated which is essential for permeability of blood vessels. Neuropeptide Y is another example of peptide which acts as both endocrine and paracrine factor there by controlling the adipogenesis and obesity.

Resistin is a specific adipokine, which is defined "as a novel angiogenic factor which directly stimulates the proliferation of endothelial cells, migration, and the tube formation[53]".

Insulin Growth Factor-1 is a potent angiogenic factor for numerous cells and plays a critical role in regulating the integrity of vessels in the fatty tissue. IL-8 is another pivotal factor for adipocytes for maintaining the regulation of angiogenesis. Angiogenic factors includes VEGF-B, VEGF-C, and angiogenin that have been positively correlated with body mass index. [54,55]

Adiponectin, an angiogenesis inhibitor, accumulates in the circulation of lean people and safe guards against the disease like diabetes and atherosclerosis. [56]Adiponectin levels in the blood have a negative relationship with BMI and are reduced in obese humans, suggesting a negative role of adiponectin in the regulation of adipogenesis. [57]It inhibits the proliferation of endothelial cell and its survival by the activation of caspase mediated endothelial cell apoptosis[58].

Angiogenesis inhibitors which are produced endogenously - endostatin, thrombospondin 1 (TSP-1), soluble VEGFR-2 are at increased levels in obese subjects when compared to lean individuals [59,60]. PlGF, indirectly inhibits the angiogenesis of adipose tissue by negatively modulating the angiogenic activity of VEGF.

TNP-470 in obese mice, results in a reversible weight reduction and also cause reduction of fatty tissue without significantly affecting the food intakes. Other anti angiogenic factors like as angiostatin and endostatin target the cells of endothelium and not the other cell types, the anti-obesity activity of this sort of inhibitors is achieved through their antiangiogenic activity. Remarkable vascular remodeling is achieved by the angiogenesis inhibitor by stimulation of apoptotic cells and decreasing the endothelial cells which are proliferating vigorously. Interesting finding is that these anti angiogenic factor like TNP-470 also increases the sensitivity of insulin in obese animals, implying that these angiogeneic inhibitor normalizes the sensitivity of insulin and also prevents the development of type II diabetes mellitus.

PATHOGENESIS OF MICRO VASCULAR COMPLICATION OF DIABETES:

1. By the polyol pathway, there is increased flux of glucose and other sugars.

2. Increased advanced glycated end products (AGEs), intracellularly.

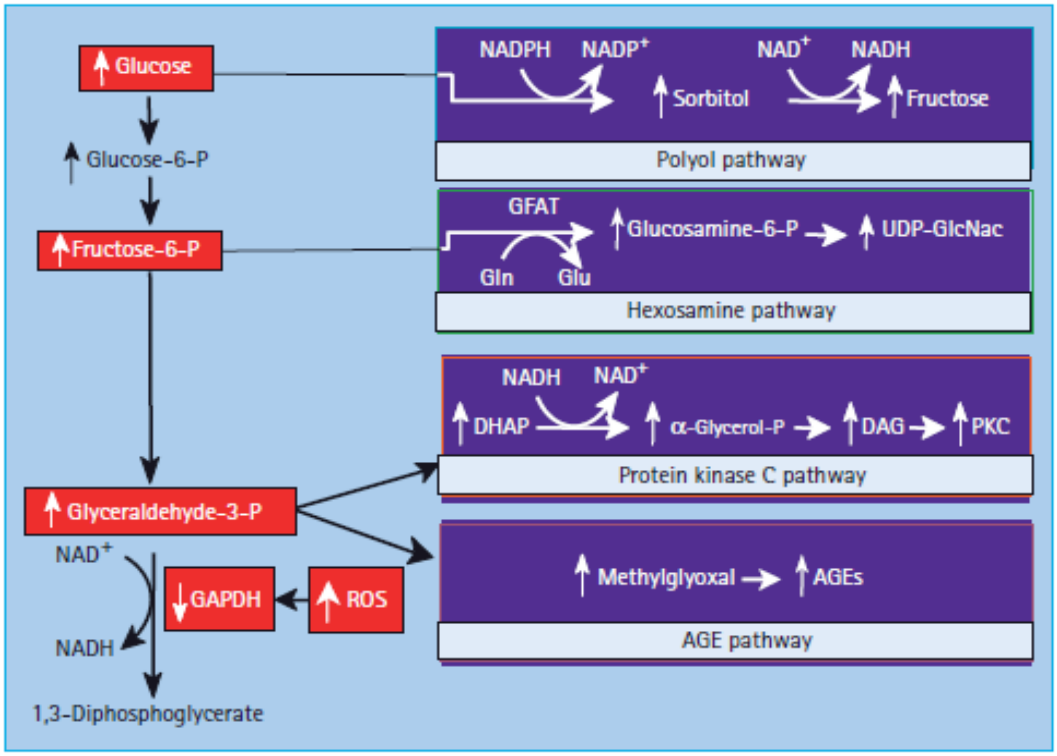
3. More production of the receptor for Advanced Glycated End products (RAGE) and its activating ligands.

4. Activation of protein kinase C (PKC) isoforms.

5. Overactivity of hexosamine pathway and a unifying hypothesis for the pathogenesis of diabetic complications is overproduction of the reactive oxygen species (ROS) superoxide by mitochondria .

When concentration of glucose is normal, aldose reductase decreases the toxic aldehydes which is generated by reactive oxygen species (ROS) to inactive alcohol but not in hyperglycemia, it can also reduce glucose to sorbitol which induces the osmotic stress by decreasing the cytosolic Na/K^{+} - ATPase activity,increased cytosolic $NADH/NAD.^{+\cdot}$ Intracellular accumulation of sorbitol results in osmotic damage in diabetic vessels. Increased sorbitol amount alters the redox

potential, elevates osmolality of cells, produces more reactive oxygen species, thereby causing cellular dysfunction.

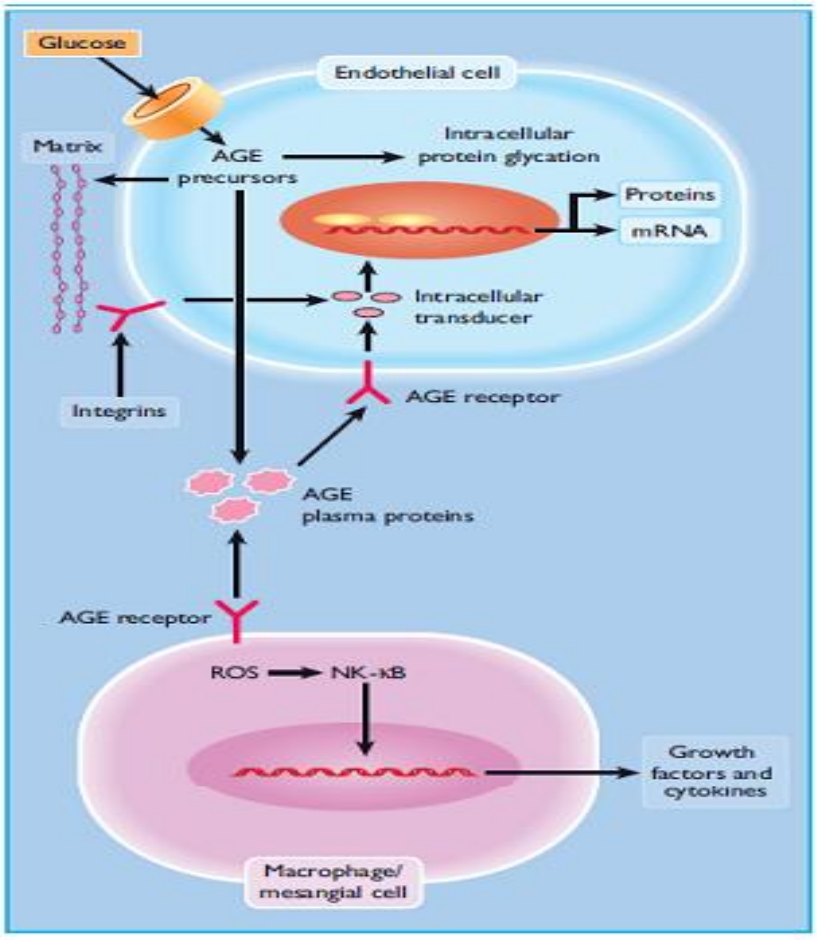


MAJOR PATHWAYS OF HYPERGLYCEMIC DAMAGE

INCREASED INTRACELLULAR ADVANCED GLYCATED END PRODUCTS

AGEs are formed by the reaction of glucose with proteins and, to nucleic acids. These reactions are reversible initially and give rise to early glycation products. Eventually they become irreversible which impair the structural and enzymatic functions of the proteins. AGEs are found in increased amounts in extracellular structures of diabetic retinal vessels [74] and renal glomeruli. [75,76,77]

AGEs can arise intracellularly by the oxidation of glucose to glyoxal [78], the conversion of an Amadori product in to the 3 deoxyglucosone, or the fragmentation of glyceraldehyde - 3 - phosphate to yield methylglyoxal [79]. All these intracellular dicarbonyls react with uncharged amino groups of intracellular proteins to form AGEs. The major intracellular AGE precursor is Methylglyoxal[80,81]



ROLE OF AGE IN MODULATING INTRACELLULAR AND EXTRA CELLULAR PROTEINS AND ITS INTERACTION OF REACTIVE OXYGEN SPECIES

INTRACELLULAR EFFECTS OF AGEs

AGE precursors can damage cells by 3 process:

1. AGE modifies the intracellular proteins which will have varied function.

2. Extracellular matrix components are modified by AGE precursors which interacts differently with other components in the matrix .

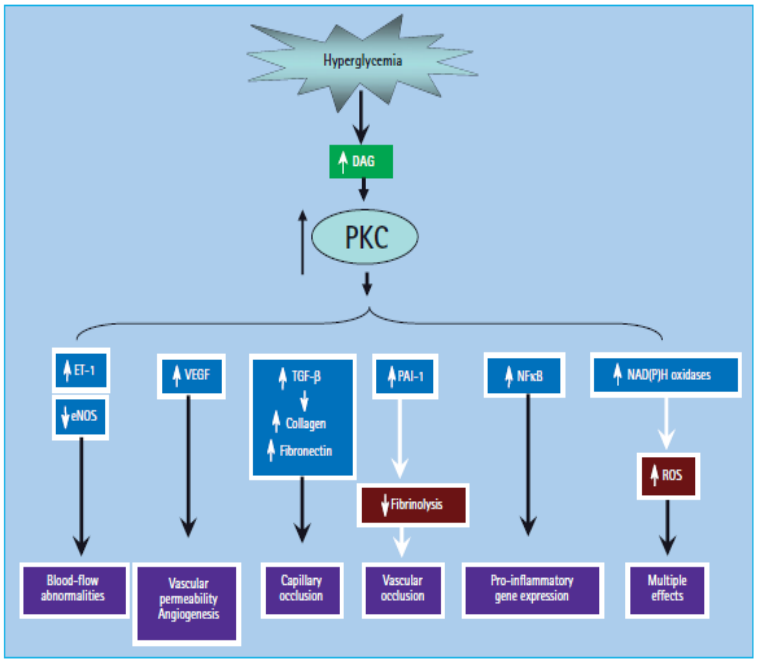
3. Plasma proteins are modified by advanced glycated end products precursors and they bind to advanced glycated end product receptors on cells like macrophage and stimulates the production of ROS, which further activates the transcription factor, nuclear factor κ B (NF κ B), resulting in numerous pathologic changes in gene expression. [82]AGE modification of intracellular protein is involved in diabetic retinopath. In diabetes, retinal capillary formation is maintained by complex interactions among pro- and anti- angiogenic factors [83,84], including angiopoietin- 2 (Ang-2).[85]

EFFECTS OF AGES ON EXTRACELLULAR MATRIX

AGE production changes the functional activities of many matrix molecules like collagen which along with AGEs were shown to form covalent intermolecular bonds which is mediated by H_2O_2

production[86] ,glycated type I collagen cross - linking results in expansion of molecular packing [87], glycated type IV collagen inhibits the normal lateral association of the molecules. AGE formation on laminin prevents it from self - assembling into a polymer. AGE induced cross - links alter the tissue function, especially in blood vessels also reduces the elasticity in arteries in diabetes. AGEs induced cross-linked proteins like collagen, extracellular matrix proteins, aggravates the process of atherosclerosis, stimulates the dysfunction of glomerulus, decreases the synthesis of nitric oxide, accelerates the dysfunction of endothelium, and composition of matrix is altered. The serum level of advanced glycated end products accumulate when GFR declines.

PATHOLOGICAL CONSEQUENCES OF PROTEIN KINASE ACTIVATION



Hyperglycemia raises the production of diacylglycerol which leads to stimulation of Protein Kinase C (PKC) which changes and modulates the transcription of genes for fibronectin, type IV collagen, contractile proteins

In diabetic proliferative retinopathy , Vascular Endothelial Growth Factor A (VEGF-A) increases and its level decreases after laser photocoagulation.

DIABETIC RETINOPATHY:

"Diabetic retinopathy is a sight-threatening chronic micro vascular complication". Diabetic retinopathy leads to a progressive changes in the retinal micro vessels which is gradual, resulting in nonperfusion of some areas in retina, increased vascular permeability leading to macular edema, abnormal proliferation of retinal vessels.

In developed countries of the world, diabetic retinopathy is the leading cause of new-onset blindness in working-aged person. More than 90% of vision loss occuring due to proliferative diabetic retinopathy can be prevented.

PREVALANCE IN WORLD

An international study states that the duration of diabetes, poor glycemic and blood pressure control are strongly associated with a higher prevalence of diabetic retinopathy. The research was presented in 2011 Annual Meeting of the Association for Research in Vision and Ophthalmology.

DISEASE	PREVALANCE IN MILLIONS (2010)	2030
DIABETIC RETINOPATHY	100.8	154.9
PROLIFERATIVE DR	20.6	31.7
DIABETIC MACULAR EDEMA	21.3	32.8
VISION THREATENING DR	33.4	51.3

World Health Organization states that the combined diabetic population of India and China is 52.4 million. This data will increase to 121.8 million and thrice of current world prevalence. Two large Indian clinical study were performed and announced the prevalence of diabetic retinopathy in southern region of India as 34.1% and 37%. Prevalence in urban South Indians was 17.6%, which was much lower than in other groups. Out of 31.7 million diabetic people who live in India, greater than 5.6 million subjects suffer from diabetic retinopathy.

MODIFIABLE RISK FACTORS FOR DIABETIC RETINOPATHY

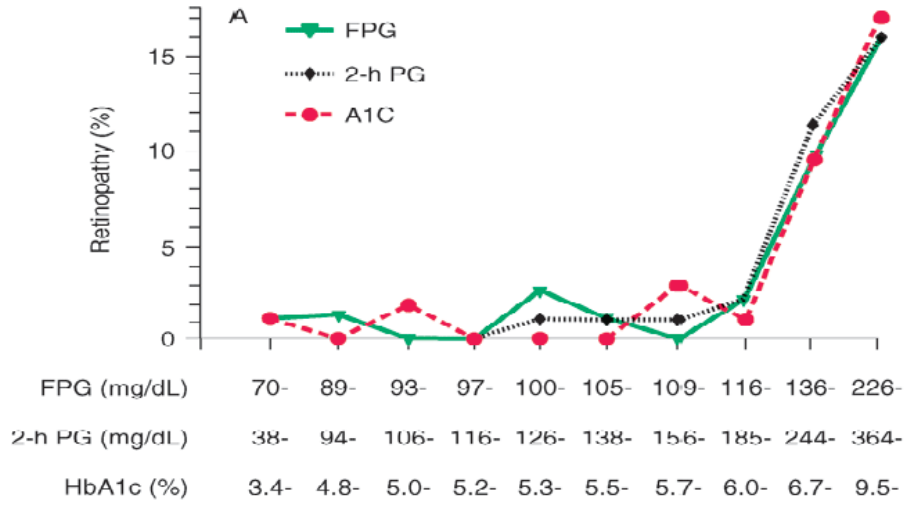
BLOOD GLUCOSE:

Tight blood glucose control decreases the risk of new onset diabetic retinopathy and decreases the progression of previously existing DR in patients with type 1 diabetes mellitus (T1DM) according to the study conducted by DCCT.[61]The UK Prospective Diabetes Study (UKPDS) [62] concluded the tight blood glucose control decreased the risk of new onset diabetic retinopathy and slows down the progression of previously occurred diabetic retinopathy in T2DM.

SYSTEMIC HYPERTENSION

Tight control of blood pressure resulted in reduction of new onset DR and slowed the progression of already existing DR. [63,64] .

CORRELATION OF DIABETIC RETINOPATHY WITH FASTING. POST PRANDIAL AND HBA1c



SERUM LIPIDS

Raised serum lipids were related with exudates at macula and moderate loss of vision .[65,66] .

Smoking

Smoking may be a risk factor in the progression of DR in T1DM as detailed by Muhlhauser *et al* [67] and Karamanos *et al.* [68]

NON - MODIFI ABLE RISK F ACTORS

Duration

Non - modifiable factor that determines the progression of DR is the duration of diabetes.[69]

Age

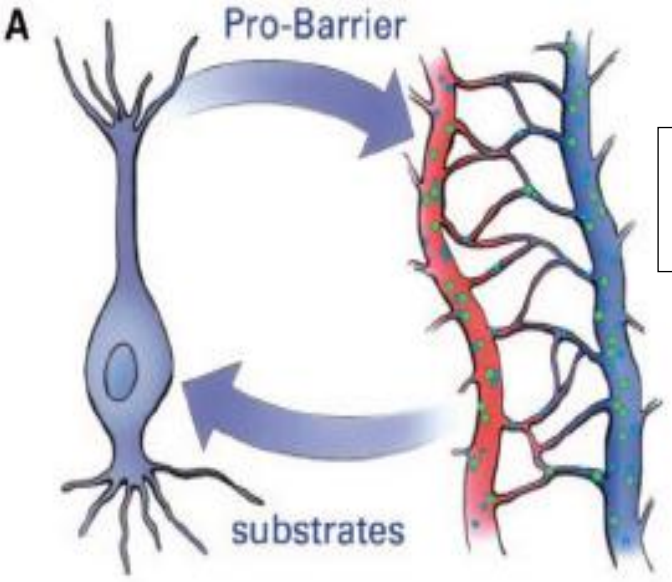
Wisconsin Epidemiological Study [70,71]stated that people who suffered from diabetes for 10 years,in the age less than 30 years, the grading and the severity was related with the older age of diagnosis where as when the age at diagnosis was 30 years or more, retinopathy severity was associated with younger age at diagnosis.

Genetic predisposition

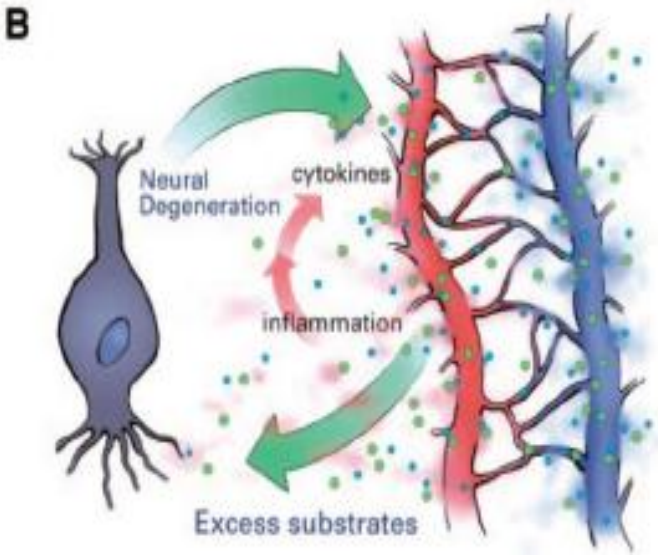
Monozygotic twins with diabetes have a strong familial clustering of DR.

Ethnicity

There was higher prevalence of DR in Latin Americans (36%) and African - Americans (29%) than for whites of Northern European ancestry (22%).[73]



NORMAL BLOOD BRAIN BARRIER

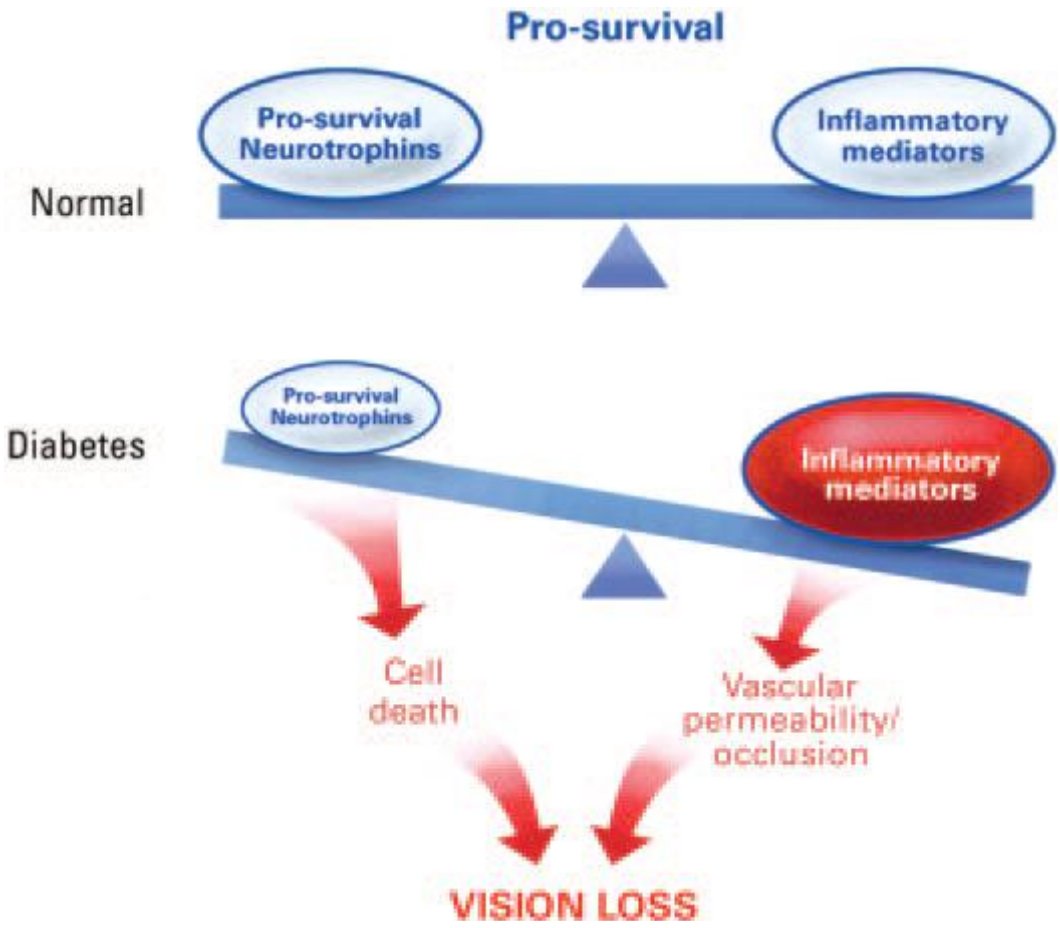


NEURO DEGENERATION LEADS TO INCREASED

GROWTH FACTORS,LOSS OF PROBARRIER

FACTORS,AGGRAVATES VASCULAR DAMAGE

ULTIMATELY LEADING TO IMPAIRED VISION



PATHOLOGICAL PHYSIOLOGICAL CHANGES IN DIABETIC RETINOPATHY

Diabetic retinopahy is microvasculopathy in which small blood vessels & retinal cells are damaged due to hyperglycemia

Cellular damage

Is due to intracellular sorbitol accumulation, oxidative oxygen free radical excess induced oxidative stress , accumulation of AGE'S and abnormal activation of protein kinase C isoforms.

Capillaropathy

Is due to death of pericytes , loss of vascular smooth muscle cells, thickening of BM and proliferation of vascular endothelial cells. Rheological changes such as abnormalities of RBC'S and WBC'S, increased platelet stickiness, and plasma viscosity may also contribute. Capillaropathy manifests with leakage and occlusion.

Neovascularization

It is due to capillary non-perfusion leading on to retinal hypoxia which results in neovascularization extending preretinally (PDR) and intraretinally; and development of intraretinal microvascular abnormalities (IRMA) are shunts that run within the retina from arterioles to venules. Neovascularisation is due to imbalance between the angiogenic(VEGF,PDGF,HGF) and anti-angiogenic (angiostatin , endostatin pigment epithelium-derived factor) .

Screening for diabetic retinopathy

All these features causes occlusion of microvessel of retina. In 1968, World Health Organization (WHO) [34] first introduced the screening tests.

SCHEDULE FOR OPTHALMIC EXAMINATION IN DIABETES

GROUP	FIRST EXAMINATION	ROUTINE FOLLOW-UP
Type 1 diabetes	Within 5 years after diagnosis of diabetes once patient is age 10 years or older	Yearly
Type 2 diabetes	At time of diagnosis of diabetes	Yearly
Pregnancy in preexisting diabetes	Prior to conception and during first trimester. Counsel on the risk of development and/or progression of retinopathy-.	Close follow-up throughout pregnancy and for one year postpartum.

NORMAL PERSON AND PATIENT WITH DIABETIC RETINOPATHY

SYMPTOMS OF DIABETIC RETINOPATHY

- Blurred vision
- Floaters
- Fluctuating vision
- Distorted vision
- Dark areas in the vision
- Poor night vision
- Impaired color vision
- Partial or total loss of vision

CLASSIFICATIONOF DIABETIC RETINOPATHY[103]:

Diabetic retinopathy is broadly classified in to two categories namely

1. Non proliferative Diabetic Retinopathy
2. Proliferative Diabetic Retinopathy

Nonproliferative Diabetic Retinopathy (NPDR)

Very mild NPDR:

Microaneurysms only

Mild NPDR

Any or all of :

- microaneurysms,
- retinal haemorrhages,
- exudates,
- cotton wool spots, up to the level of moderate NPDR.

***Moderate NPDR*:**

Any of the following:

- Severe retinal haemorrhages in 1–3 quadrants *or* mild intraretinal microvascular abnormalities (IRMA),
- Significant venous beading can be present in no more than 1 quadrant,
- Cotton wool spots commonly present.

***Severe NPDR*:**

One or more of:

- Severe hemorrhages in all 4 quadrants
- Significant venous beading in 2 or more quadrants
- Moderate IRMA in one or more quadrant

***Very severe NPDR*:**

Any two or more of criteria for severe NPDR

PROLIFERATIVE DIABETIC RETINOPATHY (PDR)

***Early PDR*:**

New vessels

- Criteria not met for high-risk PDR

***High-risk PDR*:**

- Neovascularization of the disk ≥ 1/3-1/2 disk area OR
- Neovascularization of the disk and vitreous or preretinal hemorrhage OR
- Neovascularization elsewhere ≥ 1/2 disk area AND vitreous or pretretinal hemorrhage

***Severe PDR*:**

- Posterior fundus obscured by preretinal or vitreous hemorrhage OR
- Center of macula detached

CLINICALLY SIGNIFICANT MACULAR EDEMA (CSME)

- ❖ Thickening of the retina ≤500μm from the center of the macula

OR

- ❖ Hard exudates and adjacent retinal thickening ≤500μm from macular center OR
- ❖ Zone of retinal thickening at least 1 disc area in size located ≤1 disc diameter from the center of the macula.

Microaneurysms – It is s a red spot of less than 125 micro meter

- *Small retinal hemorrhages* – It is a red spot, with irregular margins with or without uneven density, surrounding a microaneurysm.

Hard exudates:

It is a small white or yellow coloured deposits with sharp margins, seen in the in the superficial, outer layers of the retina.

Cotton wool spots

It is otherwise called as soft exudate. It is white fluffy opaque in nature. It occurs due to stasis of axoplasm in the nerve fiber layer caused by arteriolar occlusion.

***Neovascularization of the disc*:** new vessels on or within 1 disc diameter of disc margin.

Neovascularization elsewhere: new vessels elsewhere in the retina outside of disc and more than 1 disc diameter from disc margin

Diabetic retinopathy can be easily identified by complete and thorough eye examination.

Retinal imaging

Digital colour retinal photography is used to detect the lesions in retina.

Fundus fluorescein angiography

Sodium fluorescein injection is given rapidly into the arm and the fluorescence of the dye, enables photographs to be taken as it appears in the retina. The fluorescein dye cause side effects like nausea, vomiting, occasional syncope, skin rashes and itching.

Optical coherence tomography

It interprets the reflected optical waves using interferometry.

Ultrasound B scan examination

In cases of vitreous hemorrhage the presence or absence of a retinal detachment, where retinal view is obscured, can be examined.

Perimetry

It helps in the assessment of the visual field.

TREATMENT OF DIABETIC RETINOPATHY

PRIMARY PREVENTION

Strict glycemic and blood pressure control

SECONDARY PREVENTION

Annual eye examination

TERITIARY PREVENTION

ARGON LASER PHOTO COAGULATION

All eyes with clinically significant macular edema should be considered for Argon laser photocoagulation irrespective of their visual loss which is the primary therapeutic modality.

It helps in sealing of leaking micro vessels of retina and decreasing the oxygen demand in retina especially for clinically significant macula edema and high risk proliferative retinopathy

OTHER TREATMENT MODALITY

Other lasers:

- Frequency-doubled Nd:YAG laser offers the potential of a less destructive retinal effect than argon.

  The 'Pattern Scan Laser' (Pascal) uses frequency-doubled micropulse YAG in single shot mode or upto 56 shots applied in less than a second.
- Micropulse diode laser in which short duration (microseconds) burns are applied to the RPE without significantly affecting the outer retina and choriocapillaris.

Anti –VEGF agents:

Intravitreal injection of 0.5 mg ranibizumab, initially given monthly for 3 months, these agents will play an prominent role in the treatment of diabetic retinopathy in near future .

INTRAVITREAL TRIAMCINOLONE.

This can also be used with substantial results.

PARS PLANA VITRECTOMY

It is indicated in patients with

- Severe persistent vitreous haemorrhage
- Progressive tractional RD
- Combined tractional and rhegmatogenous RD
- Premacular subhyaloid haemorrhage

LIPID-LOWERING DRUGS

It may reduce the requirement for laser treatment, and studies are ongoing.

AIMS AND OBJECTIVES

AIM OF THE STUDY

PRIMARY OUTCOME:

To study the correlation of anthropometric measurements with the severity of diabetic retinopathy.

SECONDARY OUTCOME:

To study the correlation of anthropometric measurements with occult nephropathy.

To study the correlation of anthropometric measurements with serum lipids.

- Place of study: Government Royapettah Hospital, Chennai - 14 .
- Collaborative department : Department of Diabetology and Department of Ophthalmology, Government Royapettah Hospital .
- Study design: Cross sectional ,observational study.
- Sample size :100.

INCLUSION CRITERIA

- Type 1 and type 2 diabetics of all age groups were included.

EXCLUSION CRITERIA

Patients with following disease were excluded from the study.

- Hypertension
- Chronic kidney disease
- Thyromegaly
- Cervical lymph node enlargement
- Neck surgeries
- Other retinopathies
- Cataract and ocular surgeries

MATERIALS AND METHODS

METHODS AND STATISTICAL ANALYSIS

This was a clinical study of patients with type 1 and type 2 diabetes attending Government Royapettah Hospital. Ethical committee clearance was obtained from the ethical committee board of Kilpauk Medical College. Each individual signed a consent form after explaining the methodology and study purpose.

Anthropometric measurements:

Anthropometric measurements were measured using standard methods:

Weight was measured after removing slippers, and after emptying bladder using standard weighing machine. Height was measured after removing the slippers with stadiometer. Body mass index was calculated by computing the values in the formula BMI= weight in kilogram / height in metre squares. Body mass index was classified according to World Health Organisation criteria that is under weight normal weight 18-24.9%, over weight – 25-29.9% , obese > 30%. Waist circumference was measured using tape measurements at midpoint between costal margin and iliac crest. Hip circumference was measured at the level of greater trochanter and waist hip ratio were measured by dividing waist

circumference and hip circumference. Neck circumference was measured using inch tape to nearest 1 mm taken in horizontal plane below the larynx perpendicular to the long axis of neck with head straight.

Blood pressure was measured was recorded twice and average of two measurements were taken in to account. Blood samples were with drawn and serum cholesterol, triglycerides, fasting and post prandial blood sugar measured, and fundus examination was done after dilation with 1 % tropicamide and fundus grading was done by the ophthalmologist with Heine® mini ophthalmoscope . Diabetic retinopathy was graded into non proliferative (mild, moderate, severe, very severe) or proliferative (early, high risk, severe).

Statistical analysis were performed using Microsoft excel. After acquiring the complete set of data and preparing the master chart, with the help of statistician, data was analysed with Statistics Products Services Solutions (SPSS)® software for windows .Pearson correlation and Chi square test are used for finding the association of anthropometric parameters with diabetic retinopathy. p vaue <0.05 was considered to be significant and p value < 0.01 was very significant.

RESULTS

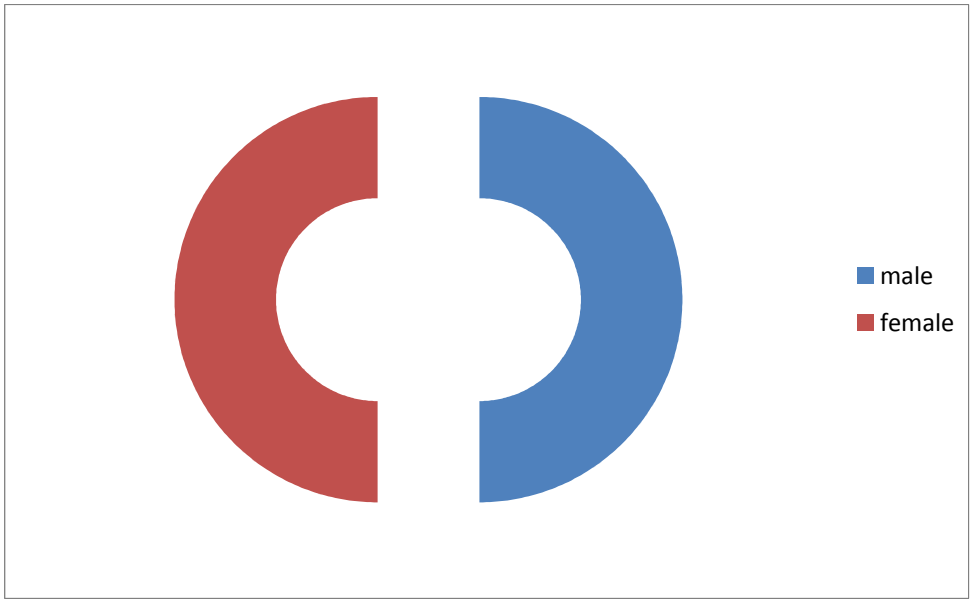
OBSERVATION AND ANALYSIS

NO. OF PATIENTS IN THE STUDY MALE AND FEMALE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	F	50	50.0	50.0	50.0
	M	50	50.0	50.0	100.0
	Total	100	100.0	100.0	

This frequency table says that equal numbers of male and female diabetic patients participated in this study .

CHART :1

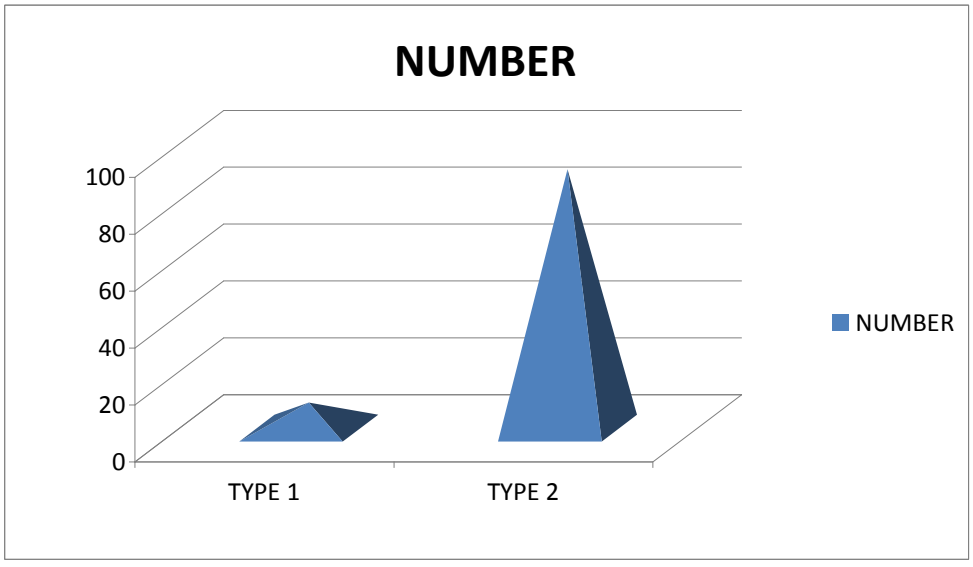


TYPE OF DIABETES IN THE STUDY POPULATION

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	9	9.0	9.0	9.0
	2	91	91.0	91.0	100.0
	Total	100	100.0	100.0	

The number of parcipatants in type 1 diabetes is 9 and in type 2 the number is 91.

CHART 2



DESCRIPTIVE ANALYSIS

	N	Range	Minimum	Maximum	Mean	Std. Deviation
AGE	100	49	19	68	51.10	9.380
DURATION	100	12	3	15	7.57	2.524
SBP	100	46	116	162	138.35	10.586
DBP	100	34	68	102	82.80	7.407
WT	100	71	35	106	67.66	12.005
HT	100	31.5	142.5	174.0	158.470	6.5958
BMI	100	23.06	17.11	40.17	26.9077	4.20264
WAIST	100	44	70	114	90.31	9.540
HIP	100	42	82	124	103.33	8.923
W/H	100	.28	.70	.98	.8691	.05659
NECK C	100	19.0	27.0	46.0	34.619	2.7067
FBS	100	257	85	342	203.17	55.793
PPBS	100	290	108	398	257.65	59.896
URINE PCR	100	.63	.01	.64	.2947	.13675
CHOLESTEROL	100	224	154	378	234.10	43.145
TGL	100	244	113	357	167.69	38.199
Valid N (listwise)	100					

Descriptive variables in the study were age, sex, duration of diabetes, blood pressure, weight, height, body mass index, waist circumference, hip circumference, waist hip ratio, neck circumference, fasting blood sugar, post prandial blood sugar, urine spot protein creatine ratio, cholesterol, triglycerides.

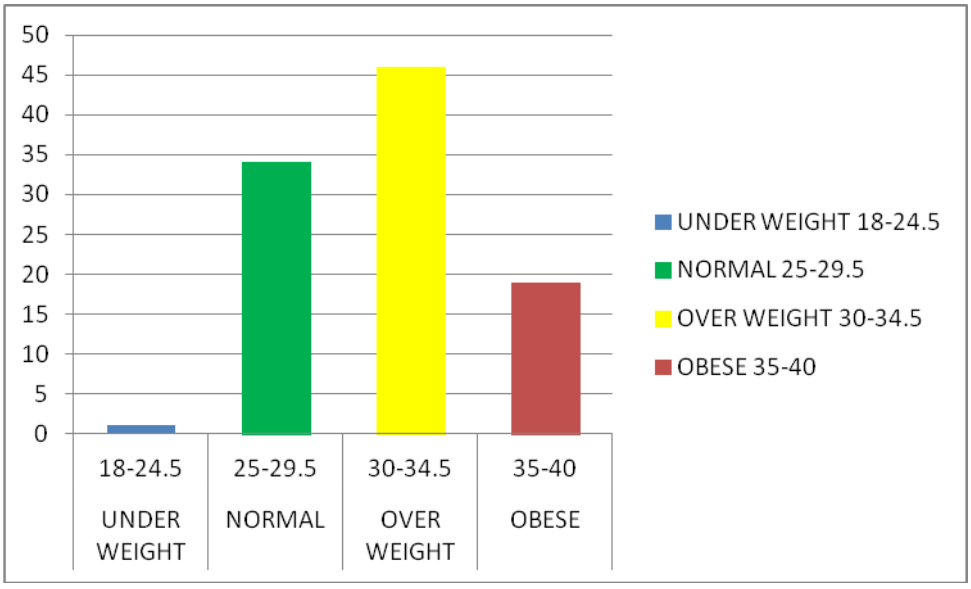
CORRELATION

		BMI	GRADE	W/H	NECK C	DURATION
BMI	Pearson Correlation	1	.235*	.213*	.466**	.354**
	Sig. (2-tailed)		.019	.034	.000	.000
	N	100	100	100	100	100
GRADE	Pearson Correlation	.235*	1	.245*	.214*	.507**
	Sig. (2-tailed)	.019		.014	.032	.000
	N	100	100	100	100	100
W/H	Pearson Correlation	.213*	.245*	1	.178	.144
	Sig. (2-tailed)	.034	.014		.077	.154
	N	100	100	100	100	100
NECK C	Pearson Correlation	.466**	.214*	.178	1	.231*
	Sig. (2-tailed)	.000	.032	.077		.021
	N	100	100	100	100	100
DURATION	Pearson Correlation	.354**	.507**	.144	.231*	1
	Sig. (2-tailed)	.000	.000	.154	.021	
	N	100	100	100	100	100

BMI_INTERVAL

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Under weight	1	1.0	1.0	1.0
	Normal	34	34.0	34.0	35.0
	overweight	46	46.0	46.0	81.0
	obese	19	19.0	19.0	100.0
	Total	100	100.0	100.0	

BMI is classified according to various class intervals .i. e 1) under weight -<18 % 2) normal weight 18- 24.9% , 3) over weight – 25-29.9% ,4) obese > 30%.



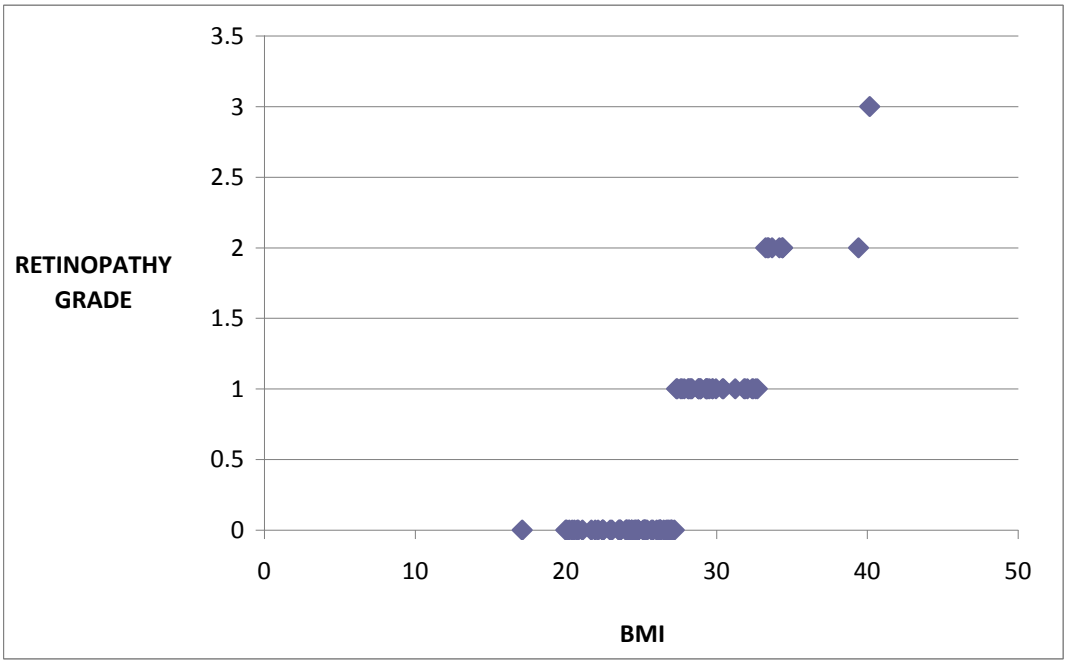
RETINOPATHY GRADE VS BMI

Pearson Correlation	.235*
Sig. (2-tailed)	.019
N	100

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

There is a positive correlation between retinopathy grade and body mass index with a significant probability value of 0.019.



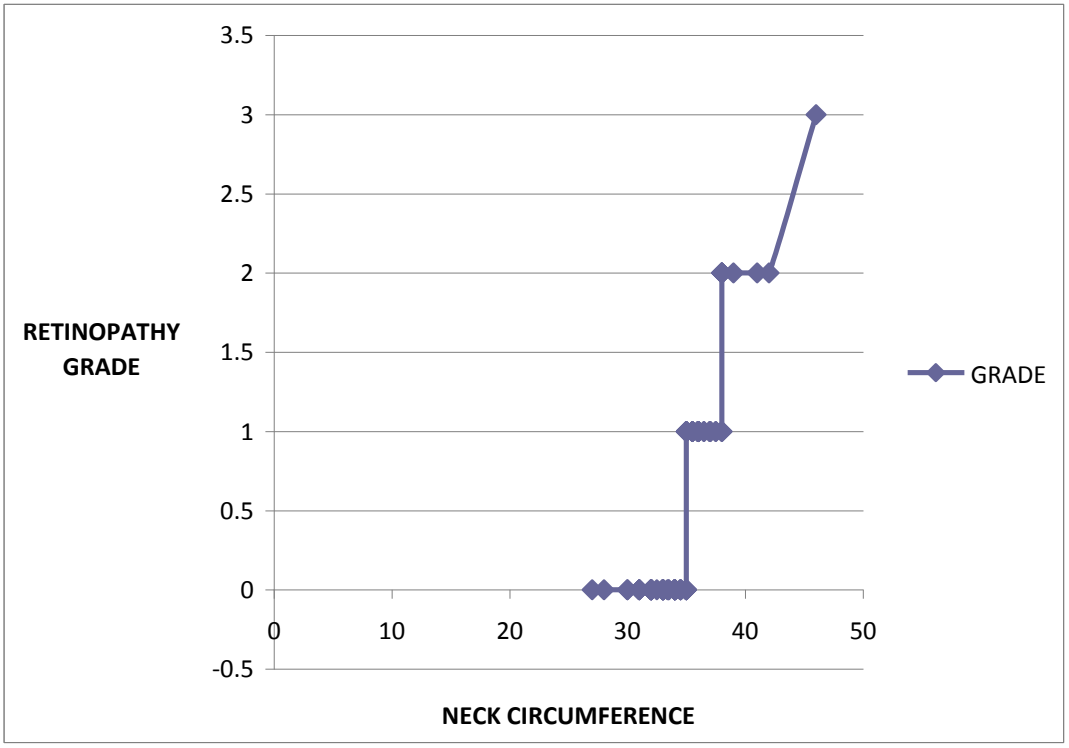
RETINOPATHY GRADE VS NECK CIRCUMFERENCE

NECK C	Pearson Correlation	.466**
	Sig. (2-tailed)	.000
	N	100

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

There is statistical significant correlation of neck circumference with retinopathy grade.



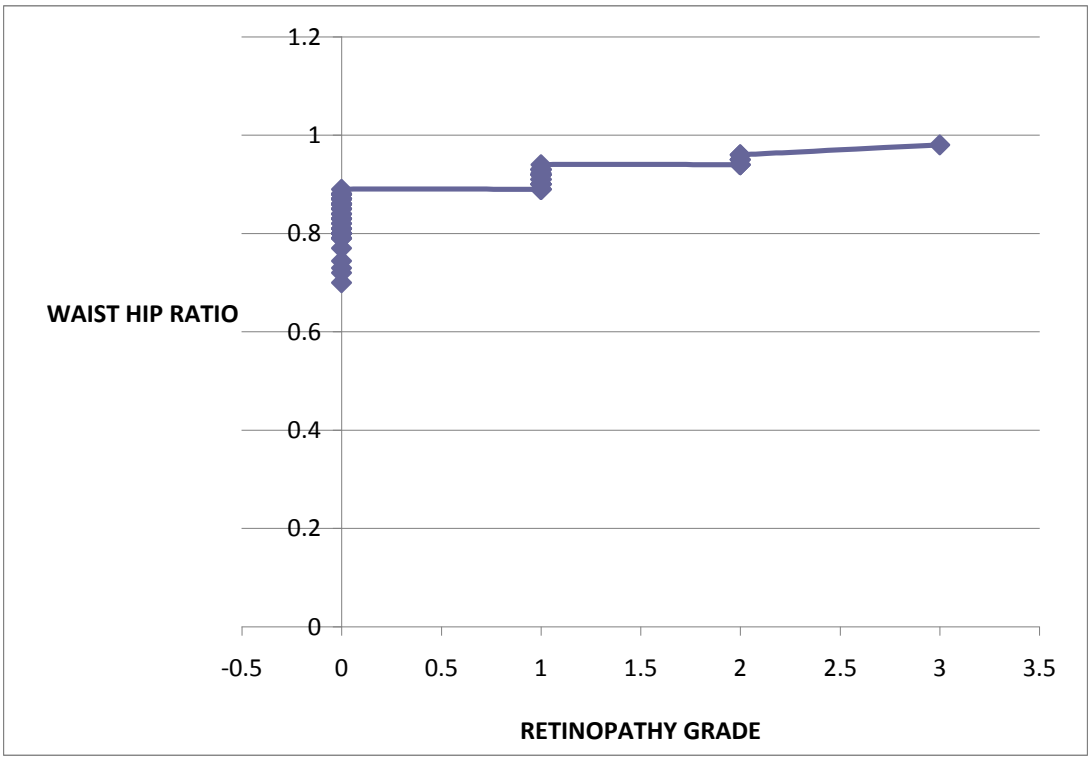
FUNDUS GRADE VS WAIST HIP RATIO

W/H	Peoarson Correlation	.245*
	Sig. (2-tailed)	.014
	N	100

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

This study shows very significant correlation of Waist hip ratio with retinopathy grade with a p value of 0.014.



SECONDARY OUTCOME:

Correlation of neck circumference and BMI vs Fundus grade, Waist hip ratio, duration of diabetes.

Correlations

			NECK C	DURATION
Spearman's rho	BMI	Correlation Coefficient	.456**	.323**
		Sig. (2-tailed)	.000	.001
		N	100	100
	GRADE	Correlation Coefficient	.494**	.409**
		Sig. (2-tailed)	.000	.000
		N	100	100
	W/H	Correlation Coefficient	.213*	.215*
		Sig. (2-tailed)	.034	.032
		N	100	100
	NECK C	Correlation Coefficient	1.000	.246*
		Sig. (2-tailed)	.	.014
		N	100	100
	DURATION	Correlation Coefficient	.246*	1.000
		Sig. (2-tailed)	.014	.
		N	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

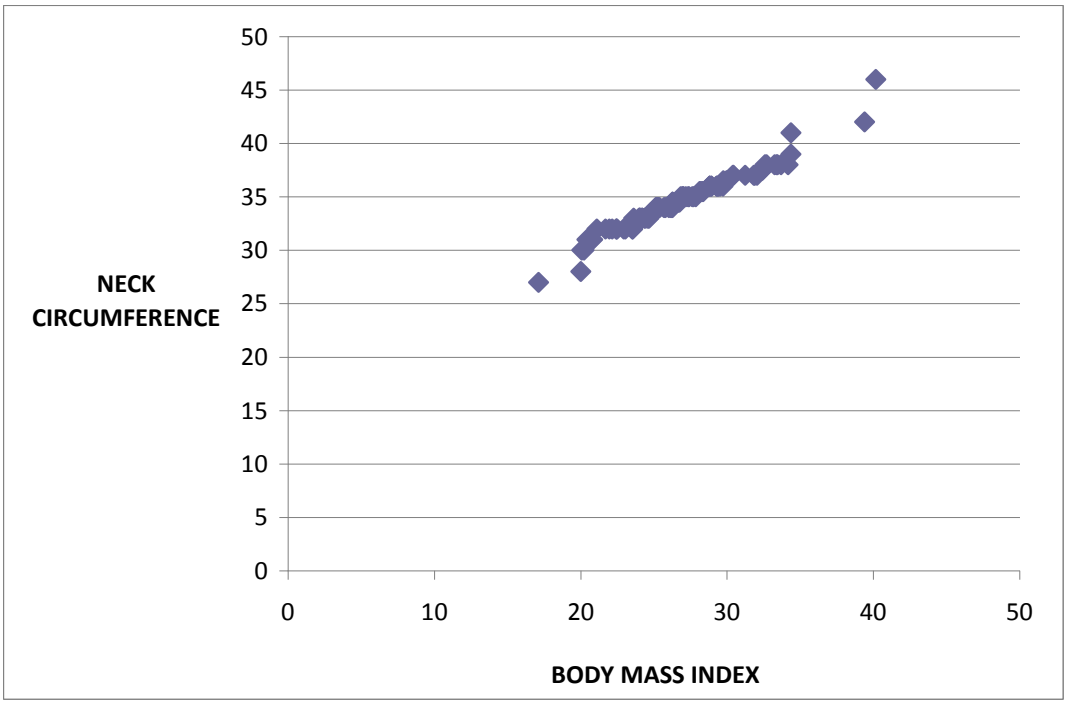
BMI VS NECK CIRCUMFERENCE

BMI	Pearson Correlation	.466**
	Sig. (2-tailed)	.000
	N	100

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

There was significant correlation between BMI and neck circumference with the p value significant (p< 0.001).



BMI VS WAIST HIP RATIO

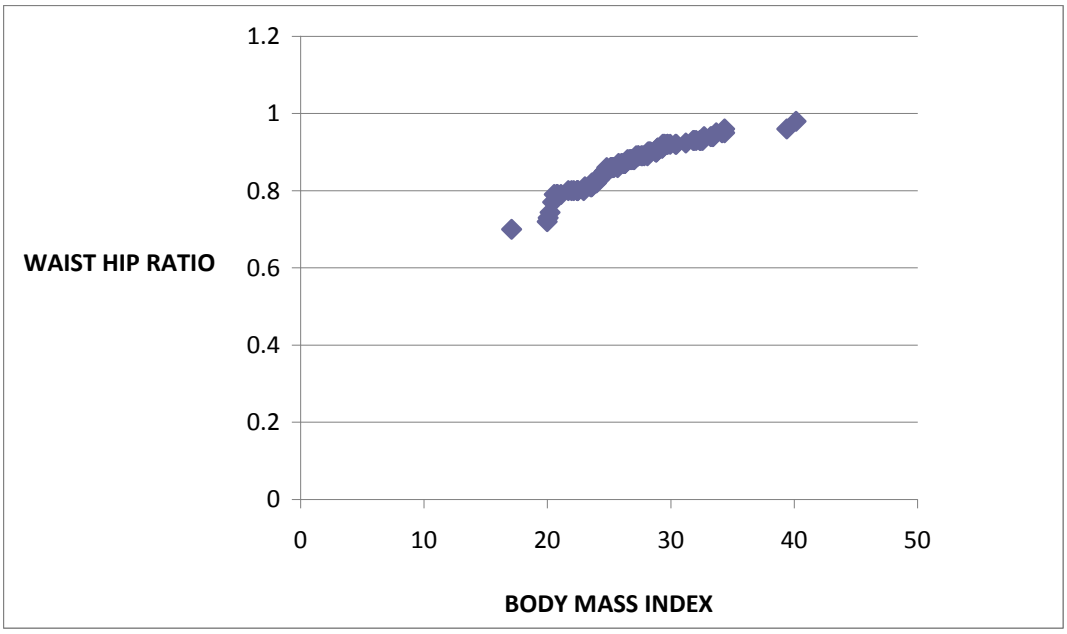
W/H	Pearson Correlation	.213*
	Sig. (2-tailed)	.034
	N	100

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

This table shows the linear correlation of BMI and waist hip ratio with a significant probability value of 0.034.

Correlation of body mass index and waist hip ratio



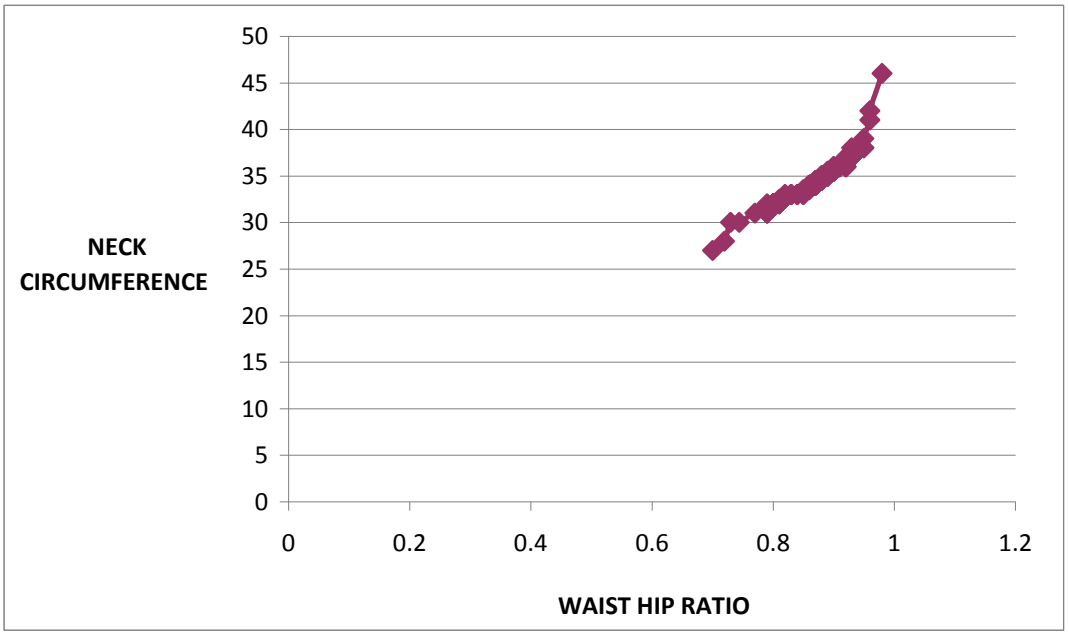
NECK CIRCUMFERENCE VS WAIST HIP RATIO

NECK C	Pearson Correlation	.178
	Sig. (2-tailed)	.077
	N	100

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

The correlation of neck circumference and waist hip ratio was not statistically significant.



DURATION VS FUNDUS GRADE

GRADE	Pearson Correlation	.507**
	Sig. (2-tailed)	.000
	N	100

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

This study showed a positive correlation of duration of diabetes with retinopathy grade with the significant p value <0.001.

BMI VS FUNDUS GRADE IN FEMALES

		BMI_interval				
		1.00	2.00	3.00	4.00	Total
GRADE	0	1	14	13	3	31
	1	0	1	7	6	14
	2	0	1	2	1	4
	3	0	1	0	0	1
Total		1	17	22	10	50

a. SEX = F

Chi-Square test

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	12.271[a]	9	.198
Likelihood Ratio	13.615	9	.137
Linear-by-Linear Association	.009	1	.925
N of Valid Cases	50		

a. 12 cells (75.0%) have expected count less than 5. The minimum expected count is .02.

b. SEX = F

In females, the body mass index does not have significant statistical correlation with the grading of diabetic retinopathy.

GRADE * BMI_interval

		BMI_interval			Total
		2.00	3.00	4.00	
GRADE	0	12	14	1	27
	1	5	9	4	18
	2	0	1	3	4
	3	0	0	1	1
Total		17	24	9	50

a. SEX = M

Chi-Square Tests[b]

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	17.990[a]	6	.006
Likelihood Ratio	16.515	6	.011
Linear-by-Linear Association	12.097	1	.001
N of Valid Cases	50		

a. 8 cells (66.7%) have expected count less than 5. The minimum expected count is .18.

b. SEX = M

In males, our study showed the positive correlation of BMI grade and fundus grade in males with p value of 0.006.

DURATION OF DIABETES VS BMI AND GRADE

DURATION	Pearson Correlation	.354**	.507**
	Sig. (2-tailed)	.000	.000
	N	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Duration of diabetes had a positive correlation with grading of retinopathy and BMI.

URINE SPOT PCR VS BMI

URINE PCR	Pearson Correlation	.208*
	Sig. (2-tailed)	.038
	N	100

Urine spot PCR had a positive correlation with body mass index with p value 0.038

TGL VS FUNDUS GRADE

TGL	Correlation Coefficient	.241*
	Sig. (2-tailed)	.016
	N	100

TGL had a direct positive correlation with diabetic retinopathy grade with p value of 0.016.

CHOLESTEROL VS NECK CIRCUMFERENCE AND BMI

CHOLESTEROL	Pearson Correlation	.302**	.305**
	Sig. (2-tailed)	.002	.002
	N	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

Cholesterol has a positive correlation with neck circumference and BMI

SYSTOLIC AND DIASTOLIC BLOOD PRESSURE VS GRADE

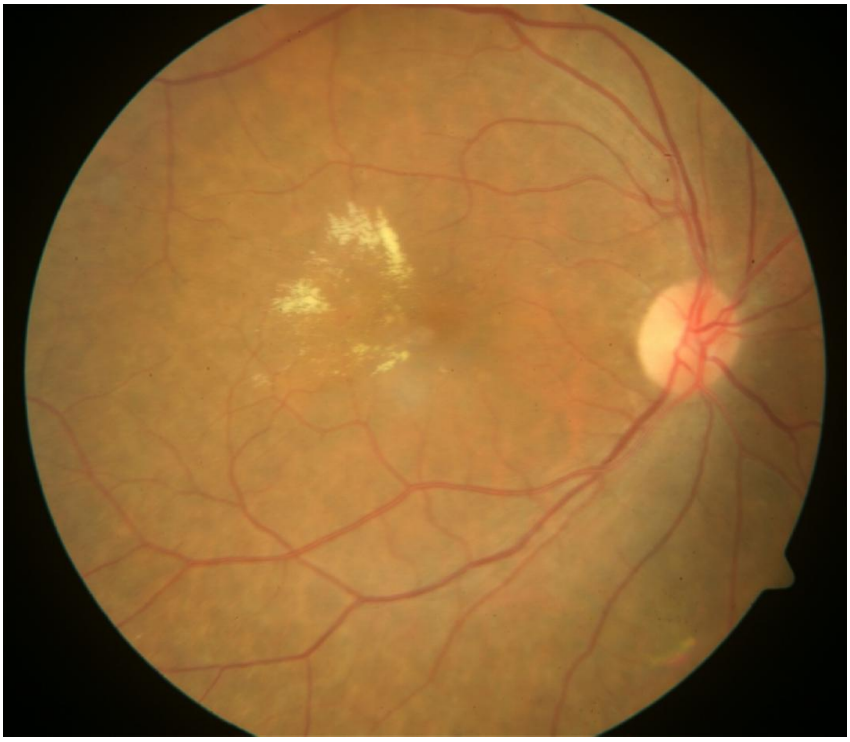
SBP	Pearson Correlation	-.117
	Sig. (2-tailed)	.247
	N	100
DBP	Pearson Correlation	-.027
	Sig. (2-tailed)	.793
	N	100

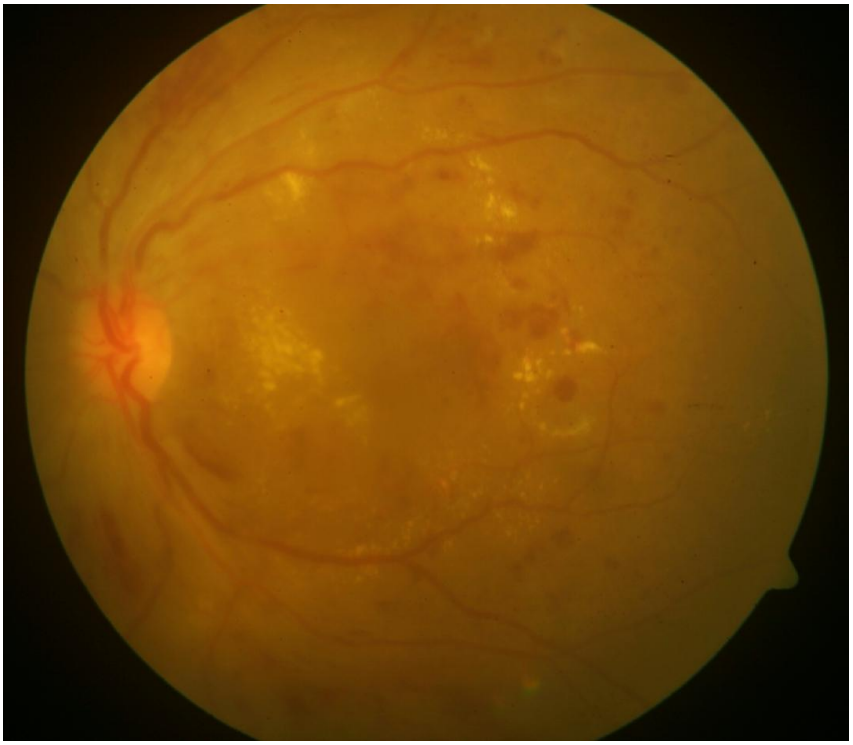
**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

In our study there was a negative correlation of systolic and diastolic blood pressure with degree of fundal retinopathy.

MILD NON PROLIFERATIVE DIABETIC RETINOPATHY WITH CSME





MODERATE NON PROLIFERATIVE DIABETIC RETINOPATHY

PROLIFERATIVE DIABETIC RETINOPATHY

DISCUSSION

DISCUSSION

Our study had equal number of male and female patients to avoid confounding factors. Type 2 diabetes patients were more in this study compared to type 1 diabetes which indirectly implies that prevalence of type 2 diabetes was more.

For more than thirty years it was thought poor glycemic control, poor blood pressure and longer duration of diabetes were only found to affect the occurrence and progression of diabetic retinopathy but later newer studies like ADVANCE [96] Action in Diabetes and Vascular Disease and the Action to Control Cardiovascular Risk in Diabetes ACCORD[97]-Eye proved that other factors too play a role in progression of diabetic retinopathy. Other modifiable risk factors has to be thoroughly studied.

Hence in our study, we aimed at studying the correlation of anthropometric changes and severity of diabetic retinopathy in the study population of hundred diabetics. This clinical study showed the positive association of body mass index and neck circumference with the severity of diabetic retinopathy in males.

BMI AND RETINOPATHY

Many studies have been conducted globally, to prove the correlation of anthropometric parameters such as body mass index with diabetic retinopathy. Diabetic Control and Complication Trial[98] showed that body mass index had positive association with the diabetic retinopathy. Another study by hoorm[99] analysed 2484 patients and concluded that there was linear correlation of diabetic retinopathy with BMI, but the draw back with that study again was they had not included other anthropometric measurements like waist, hip circumference and waist hip ratio.

Singapore study (SiMES) [100](2004- 2006) –a multivariate analysis reported that there existed a negative correlation between body mass index and diabetic retinopathy. The drawback regarding that study was that, it didn't include the educational status, duration of diabetes and economic status of the patients, limited sample size of only eighty members and only random blood sugar was used to diagnose diabetes which was obviously not the proper diagnostic test. Sankara Nethralaya Diabetic Retinopathy Epidemiology And Molecular genetic Study (SN-DREAMS-I) conducted a study in urban parts of India which comprised 30% of Indian population. With the cut off value for obesity as BMI ≥

23kg/m^2 and WC ≥ 90cm in males and ≥ 80cm in females, there was a positive relationship between abdominal obesity and increased waist hip ratio with diabetic retinopathy among females. Limitation of this study was that only BMI was taken as the index for measuring obesity but other anthropometric parameters were not included.

Wisconsin Epidemiologic Study of Diabetic Retinopathy[101] group conducted the study with thousand three hundred and seventy patients aged thirty or older and concluded that lower body mass index correlated with the severity of diabetic retinopathy and later after twenty years the WESDR group showed the positive correlation of body mass index with retinopathy in insulin dependent diabetes. Raman et al and Looker et al mentioned that lower body mass was associated with diabetic retinopathy, contributing that diabetic patients have a mind set of reducing weight once the diagnosis of diabetes is established. In 1986, Ballard et al and Boyvada et al in 2003 and Van Leiden et al in 2005 showed that increased body mass index was associated with severe diabetic retinopathy.

GENDER DIFFERENCES IN RETINOPATHY

According to study conducted by Janghorbani et al [30], gender had no role in the severity of diabetic retinopathy. Brandon et al showed that Indian women had more abdominal fat. WESDR [101]emphasized that males were more prone for severe diabetic retinopathy. In our study, female had less severity, the reason has to be evaluated with large number of population and by taking equal number of participants in underweight, normal weight, overweight, obese in both males and females.

We were able to find that most of the diabetics in our study had mild retinopathy both in normal weight and in over weight. World Health Organisation declared the healthy body mass for Asians is 18.5 -22 kg /m^2. Compared to other ethnic groups, Indians have less BMI. If the BMI interval according to Asian Indian cut off were used, more number of patients would have fallen into obese category and linearly the proportion of patients with moderate and severe retinopathy would increase. In our clinical study, amongst females, neck circumference, waist hip ratio and duration of diabetes were strongly associated with each other and with the retinopathy changes in diabetics but BMI did not have a statistically significant correlation. The reason behind this might

be that equal number of participants were not recruited in each arm of BMI interval in females. Whereas in males BMI, neck circumference and duration of diabetes were associated with each other but waist hip ratio was not found to be associated. **Urine spot PCR** had a positive correlation with body mass index and negative correlation with systolic blood pressure, diastolic blood pressure, neck circumference in this clinical study. Systolic and diastolic blood pressure did not correlate with diabetic retinopathy grade in our study. Badaruddoza et al studied relationship of waist hip ratio, BMI and blood pressure among the Punjabi Sikh and Hindu females and concluded that they had positive correlation with each other.

Advantages of measuring neck circumference:

There exists a linear correlation of waist-hip circumference, waist hip ratio, neck circumference, serum lipids with diabetic retinopathy. Hence, neck circumference could be used independently to measure central obesity or upper body fat. Also, neck circumference and serum lipid levels namely serum cholesterol and triglyceride were positively correlated, which indicates that neck circumference could be used a useful indicator for metabolic syndrome and instead of measuring weight and height and computing the formula of body mass index, neck

circumference can be easily measured. Cultural dilemma exists while measuring the waist circumference and hip circumference among the females, where neck circumference measurements would be easily acceptable and neck circumference can well be proclaimed as a surrogate marker of cardio metabolic disease. **Duration of diabetes** had positive correlation with diabetic retinopathy grade. J.L. Gross1 et al in 2006 concluded that the duration of diabetes had a positive correlation of diabetic retinopathy grade.

WAIST CIRCUMFERENCE VS WAIST HIP RATIO .

Sunita et al study reported that waist circumference was a better surrogate marker of body mass index than waist hip ratio. In the above study, they proclaimed that waist hip ratio measures the regional distribution of fat rather than the measurement of central obesity through waist circumference. According to our study, the waist hip ratio and the waist circumference goes hand in hand with body mass index and with the severity of diabetic retinopathy. Asian cut off value for normal waist circumference were 85 and 80 cm for men and women and waist hip ratio were 0.89 and 0.81 for men and women respectively.

In 2001, first cross sectional study was conducted by Ben – noun *et al*. 3 to find out any association between neck circumference and obesity, and to identify if obesity can be recognised only by measuring the neck circumference. He showed in the results that, neck circumference of greater than 37 cm and 34 cm in male and female respectively, was associated with overweight/obesity. The prevalence of diabetic retinopathy had a linear association till the BMI of 26-29.9% and for diabetic nephropathy linear correlation exist for BMI of > 30 . The Chennai Urban Rural Epidemiology Study (CURES) Eye Study—2 by M Rema et al in 2006 showed that there was a positive correlation between triglycerides and diabetic retinopathy and macular edema. Our study concluded that serum cholesterol and triglycerides had a positive correlation with diabetic retinopathy.

LIMITATION;

- The results might not be generalised to other ethnic groups due to varied cultural practices, eating habits and physical work and varying urbanisation as the study was performed in urban population.
- In this study the body mass index was classified according to the western standards.

- Neck circumference was concluded as a surrogate marker of obesity but there was no radiographic quantification measures to prove this fat distribution.
- This study included only hundred diabetic patients. Further studies with large number of sample size has to be undertaken in order to reiterate this fact.

FUTURE IMPLICATION:

- Healthy food habits, and increased physical activity has to be encouraged for it is the most important intervention in reducing the risk of future development of lifestyle disorders.
- Diabetic patients have to be screened periodically as per international/ national guidelines, to avoid the consequences of retinopathy and vision loss.
- Neck circumference is considered to be a surrogate marker of central obesity .
- Use of Angiogenesis inhibitors has to be evaluated in obesity.

CONCLUSION

CONCLUSION

From the study ,as the body mass index ,neck circumference increases, the severity of retinopathy increases in males , where as waist hip ratio does not have positive correlation with grade of retinopathy .Neck circumference has significant correlation with grade of diabetic retinopathy.

In females, the body mass index does not have statistically significant correlation with diabetic retinopathy .Neck circumference, waist cizc rcumference, waist hip ratio have positive correlation with DR. Duration of diabetes has positive correlation with diabetic retinopathy and BMI. Urine spot PCR had a positive correlation with body mass index.

Hence in diabetics anthropometric measures help in predicting the micro vascular complications such as retinopathy and nephropathy. Therefore such simple noninvasive anthropometric measures have to be undertaken routinely while following up the patients.

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ANNEXURES

ABBREVIATIONS

DM	-	DIABETES MELLITUS
DR	-	DIABETIC RETINOPATHY
IHD	–	ISHAEMIC HEART DISEASE
WHO	-	WORLD HEALTH ORGANISATION
BMI	-	BODY MASS INDEX
SBP	-	SYSTOLIC BLOOD PRESSURE
DBP	-	DIASTOLIC BLOOD PRESSURE
WT	-	WEIGHT
HT	-	HEIGHT
W/H	-	WAIST TO HIP RATIO
FBS	-	FASTING BLOOD SUGAR
PPBS	-	POST PRANDIAL BLOOD SUGAR
HBA1C	-	GLYCATED HEMOGLOBIN
TGL	-	TRIGLYCERIDES
HDL	-	HIGH DENSITY LIPOPROTEIN
LDL	-	LOW DENSITY LIPOPROTEIN
VLDL	-	VERY LOW DENSITY LIPOPROTEIN
AGE	-	ADVANCED GLYCATED END PRODUCTS
VEGFR	-	VASCULAR ENDOTHELIAL GROWTH FACTOR
IL-6	-	INTER LEUKIN 6
PAI	-	PLASMINOGEN ACTIVATOR INHIBITOR

FFA	-	FREE FATTY ACID
FGF	-	FIBROBLAST GROWTH FACTOR
MMP	-	MATRIX METALLOPROTEINASE.
PKC	-	PROTEIN KINASE C
ECM	-	EXTRA CELLULAR MATRIX
IOTF	-	INTERNATIONAL OBESITY TASK FORCE
DCCT	-	DIABETIC CONTROL AND COMPLICATION TRIAL
UKPDS	-	UK PROSPECTIVE DIABETIC STUDY

DATA COLLECTION FORM

- **NAME**
- **AGE**
- **SEX:**
- **OCCUPATION:**
- **ADDRESS:**
- **OP NO**
- **DURATION OF DIABETES, YR**
- **MEDICATIONS**
- **OCULAR SYMPTOMS**
- **CURRENT/PAST SMOKER, PACK/YEAR**
- **SYSTOLIC BLOOD PRESSURE, MM HG**
- **DIASTOLIC BLOOD PRESSURE, MM HG**
- **ECG**
- **WEIGHT, KG**
- **HEIGHT, M**
- **BODY MASS INDEX, KG/M2**
- **WAIST CIRCUMFERENCE, CM**
- **WAIST TO HIP RATIO, CM**
- **NECK CIRCUMFERENCE, CM**
- **FASTING PLASMA GLUCOSE, MG/DL**
- **POST PRANDIAL SUGAR MG/DL**
- **TOTAL CHOLESTEROL, MG/DL**
- **TRIGLYCERIDES, MG/DL**
- **RETINOPATHY GRADE**

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE.
CHENNAI-10
Ref.No.6206/ME-1/Ethics/2012 Dt:05.07.2012.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on The Correlation of anthropometric measurements with retinopathy changes in diabetes" submitted by Dr.K.Devi, MD (General Medicine), PG Student, Govt Royapettah Hospital, Chennai

The Proposal is APPROVED

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE * Kilpauk, Chennai-10. * 12 OCT 2012

12/10/12

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

MASTER CHART

NO	NAME	AGE	SEX	TYPE	YRS	SBP	DBP	WT	HT	BMI	WAIST	HIP	W/H	N C	FBS	PPBS	URINE PCR	CHOLESTEROL	TGL	FUNDUS	GRADE
1	MAHESWARI	48	F	II	7	130	76	78	156.5	31.84	97	108	0.89	37.5	220	280	0.38	230	216	MILD NPDR	1
2	VASANTHAN	62	M	II	8	140	78	88	164	32.71	90	102	0.88	35.5	246	300	0.28	278	158	MILD NPDR	1
3	SARASWATHI	54	F	II	5	149	98	67	160	26.17	82	95	0.86	34	186	226	0.01	208	156	NIL	0
4	SHANTHI	34	F	I	8	129	78	62	162	23.6	76	90	0.84	31	183	228	0.29	216	164	NIL	0
5	MURUGESAN	42	M	II	5	128	72	78	168	27.63	86	92	0.93	34	218	268	0.32	222	167	NIL	0
6	KUTTIAMAL	52	F	II	8	116	70	64	156	25.4	84	102	0.82	33.5	178	190	0.34	176	142	NIL	0
7	SUBRATHA	42	F	II	4	134	82	60	149	27.02	81	96	0.84	31	162	188	0.18	186	161	NIL	0
8	KALA	52	F	II	7	146	84	68	152	29.43	98	106	0.92	36	190	285	0.42	207	232	MILD NPDR WITH CSME	1
9	VALLI	38	F	I	10	138	86	66	162	28.95	89	98	0.9	34.5	219	239	0.32	243	156	MILD NPDR	1
10	SELVAM	46	M	II	6	126	84	66	156.5	26.94	86	96	0.89	33	218	256	0.07	221	172	NIL	0
11	MUTHUMEENA	63	F	II	12	136	78	83	157.5	33.45	110	118	0.93	35.5	224	288	0.33	234	179	MOD NPDR	2
12	BANUMATHI	40	F	II	6	128	76	70	154.5	29.32	89	110	0.8	35	154	312	0.54	264	168	NIL	0
13	NADHA	56	M	II	7	134	84	67	156.5	27.35	97	102	0.95	35.5	187	195	0.21	258	150	MILD NPDR	1
14	SAKUNTHALA	48	F	II	5	126	70	59	158	23.63	84	102.5	0.81	33.5	232	254	0.26	225	192	NIL	0
15	ALI	45	M	II	8	132	84	78	166	28.3	98	107	0.91	37	214	326	0.28	211	134	MILD NPDR	1
16	SARAVANAN	56	M	II	7	144	88	60	158	24.03	96	106	0.9	38	342	365	0.47	378	198	MILD NPDR	1
17	VIJAYAN	36	M	I	8	132	76	58	159	22.94	87.9	93	0.94	33.4	217	253	0.29	214	172	nil	0
18	GOWRI	45	F	II	5	138	84	56	146	26.27	78	98	0.79	32	256	298	0.21	256	113	nil	0
19	NOORNISHA	49	F	II	6	152	84	78	156	32.05	89	104	0.85	34	238	267	0.31	258	146	MILD NPDR	1
20	LAKSHMI	54	F	II	6	132	88	66	160	25.78	92	116	0.79	33	254	267	0.12	276	150	NIL	0
21	MAIKAMMAL	62	F	II	8	126	84	67	154	28.25	94	102	0.92	32	264	306	0.39	312	143	MILD NPDR	1
22	HEMAVATHI	59	F	II	9	132	68	62	156.5	25.31	96	102	0.94	34	247	284	0.31	267	152	NIL	0
23	MANIKKAM	51	M	II	7	138	82	72	158	28.84	88	106	0.83	36	183	290	0.37	269	156	NIL	0
24	JAYAKANDHAM	35	F	I	5	142	84	56	158	22.43	93	116	0.8	32	182	278	0.11	284	149	NIL	0
25	SARUMATHY	64	F	II	9	139	76	54	162	20.57	85	98	0.86	32	173	265	0.42	178	135	NIL	0
26	SUGIRITHA	49	F	II	4	146	88	64	162	24.38	78	89	0.87	37	263	252	0.18	167	152	NIL	0
27	SHIVA	53	M	II	8	148	90	80	153	34.17	89	106	0.83	36	257	306	0.04	285	187	MILD NPDR	1
28	DHALAYAN	59	M	II	6	134	82	74	161.5	28.37	92	118	0.77	35	276	261	0.3	276	205	NIL	0
29	VANI	48	F	II	9	128	78	68	166	24.67	98	108	0.9	36.5	303	349	0.21	243	184	MOD NPDR	2
30	GAUTHAMAN	52	M	II	12	156	72	72	149	32.43	109	118	0.923	39	296	379	0.38	278	254	SEVERE PDR	3
31	KUPPAN	56	M	II	8	150	82	78	162	29.72	92	104	0.88	36	218	265	0.16	244	156	NIL	0
32	SEKAR	47	M	II	5	146	76	66	160	25.7	88	99	0.88	33	176	245	0.34	215	188	NIL	0
33	AHJA MOIHEED	58	M	II	9	160	82	56	158	22.43	84	103	0.81	35	153	178	0.21	220	156	NIL	0
34	IYAPPAN	30	M	I	10	126	74	72	158	28.84	94	114	0.82	36	198	267	0.24	284	139	MILD NPDR	1
35	VASU	59	M	II	6	138	82	58	170	20	86	95	0.9	34.5	278	315	0.54	236	132	NIL	0
36	USMAN	63	M	II	9	142	88	70	164	26.02	90	98	0.91	36	317	386	0.37	167	183	MILD NPDR	1
37	VADIVEL	28	M	I	8	118	68	62	158	24.8	79	88	0.89	30	249	256	0.11	234	115	MILD NPDR	1
38	GUNALAN	54	M	II	9	140	72	69	159	27.2	102	118	0.86	35	252	267	0.27	167	122	NIL	0
39	VINCET	26	M	I	6	126	72	58	157	23.53	95	106	0.89	32	152	179	0.17	189	149	MILD NPDR	1
40	BALU	54	M	II	8	156	84	73	161	28.16	99	111	0.89	33	178	190	0.55	278	196	NIL	0
41	GANESAN	48	M	II	7	142	88	59	155	24.55	83	96	0.86	31	277	343	0.63	163	143	NIL	0
42	MURUGAVEL	45	M	II	5	136	72	66	162	25.14	79	89	0.88	32	318	356	0.44	182	256	MILD NPDR	1
43	ELUMALAI	62	M	II	4	124	70	61	159	24.12	85	94	0.9	34	246	289	0.51	299	357	NIL	0
44	BALAJI	43	M	II	6	132	82	64	156	26.29	93	106	0.87	35	215	267	0.32	321	253	NIL	0
45	MANIVEL	59	M	II	10	148	80	72	158	28.8	80	109	0.73	34.5	283	346	0.18	216	217	NIL	0
46	VANI	46	F	II	11	156	76	88	160	34.37	82	95	0.86	34	166	189	0.13	256	137	MILD NPDR	1
47	GOWRI	61	F	II	5	162	92	76	162	28.95	93	106	0.87	33	153	168	0.01	214	158	NIL	0
48	KUMAR	54	M	II	8	154	86	82	157	33.26	84	98	0.85	35	178	215	0.23	175	177	NIL	0
49	KALYANI	47	F	II	6	148	74	76	158	30.44	82	94	0.87	34	122	162	0.43	269	209	MILD NPDR	1
50	KASTHURI	49	F	II	9	132	82	72	155	29.96	94	102	0.92	37	287	365	0.29	283	156	MILD NPDR	1

NO	NAME	AGE	SEX	TYPE	YRS	SBP	DBP	WT	HT	BMI	WAIST	HIP	W/H	N C	FBS	PPBS	URINE PCR	CHOLESTEROL	TGL	FUNDUS	GRADE
51	NAVEEN THARABAI	60	F	2	5	130	80	60	143	29.3	102	114.5	0.89	36.5	181	240	0.4	230	298	MODERATE NPDR	2
52	RAJESHWARI	54	F	2	6	142	84	60	154	25.2	110	118	0.93	35.5	109	226	0.21	278	138	MILD NPDR	1
53	LAKSHMI	66	F	2	5	148	88	65	146	30.4	105	107	0.98	33	128	233	0.28	189	162	NO EVIDENCE	0
54	LALITHA	68	F	2	8	150	90	50	142.5	24.62	73	98	0.744	32	138	186	0.18	184	152	NO EVIDENCE	0
55	PONNAMMAL	55	F	2	7	144	92	53	150	23.55	76	95	0.8	33	136	244	0.01	218	178	NO EVIDENCE	0
56	MYMUBEE	54	F	2	6	138	88	86	164	31.9	98	112	0.87	35	116	216	0.26	278	178	MILD NPDR	1
57	SELVAM	45	M	2	8	148	94	72	164	26.76	96	108	0.88	36	184	214	0.41	220	148	NO EVIDENCE	0
58	AMEENA	55	F	2	4	118	78	48	152	20.77	78	92	0.84	32.5	219	254	0.28	190	130	NO EVIDENCE	0
59	ELANGO	49	M	2	9	136	88	72	165.5	26.28	86	92	0.93	37	136	199	0.17	188	152	MILD NPDR	1
60	DHANASEKAR	56	M	2	12	158	102	92	166	33.38	103	115	0.89	35	88	188	0.29	254	116	MILD NPDR	1
61	ALAMELU	56	F	2	15	126	82	60	158	24.03	93	103	0.9	33	194	248	0.36	191	142	MILD NPDR	1
62	AFRAS	58	M	2	5	144	92	62	164	23.05	98	103	0.95	34.5	198	214	0.16	232	186	NO EVIDENCE	0
63	SULOCHANA	40	F	2	7	136	80	56	159	22.15	84	90	0.93	32	116	168	0.28	308	178	NO EVIDENCE	0
64	SAROJA	45	F	2	5	154	92	58	146.5	27.02	94	102	0.92	33	188	246	0.33	189	148	NO EVIDENCE	0
65	RAJA	62	M	2	6	138	86	70	158.5	27.86	107	118	0.9	38	198	216	0.19	228	179	MILD NPDR	1
66	MARIAMMAL	38	F	2	4	126	82	48	154	20.23	72	88	0.81	28	156	233	0.44	176	134	NO EVIDENCE	0
67	AMARAVATHI	68	F	2	6	144	86	60	150	26.66	102	119	0.85	38	216	284	0.18	258	184	MILD NPDR	1
68	KARUPPAYI	45	F	2	7	142	82	78	162.5	29.53	95	101	0.94	37	85	147	0.35	188	142	NO EVIDENCE	0
69	MANNIKKAM	60	M	2	10	160	100	88	172	29.74	96	104	0.92	35.5	186	248	0.28	287	153	MILD NPDR	1
70	SHERIFF	52	M	2	12	130	88	106	164	39.41	98	118	0.83	38	225	297	0.45	264	202	MODERATE NPDR	2
71	CHANDRAN	49	M	2	8	126	84	62	160	24.21	82	102	0.8	33	210	266	0.28	247	127	NO EVIDENCE	0
72	BASHA	58	M	2	15	138	92	82	172	27.71	86	108	0.79	36	232	398	0.62	302	256	MODERATE NPDR,CSME	2
73	SUBATHRA	56	F	2	9	140	86	78	158	31.24	103	116	0.88	38	128	212	0.48	234	158	NO EVIDENCE	0
74	SELVAMANI	48	M	2	7	148	92	68	164	25.28	88	102	0.86	33	188	246	0.35	278	178	NO EVIDENCE	0
75	PREMA	64	F	2	3	146	94	51	158	20.42	84	103	0.81	34.5	214	324	0.29	188	124	NO EVIDENCE	0
76	CHANDRAVADHANAM	62	M	2	8	138	88	82	156	33.69	96	114	0.83	36	320	356	0.52	294	182	MILD NPDR	1
77	PRIYA	19	F	1	3	118	80	35	143	17.11	71	82	0.86	27	92	108	0.18	174	132	NO EVIDENCE	0
78	MANIVANNAN	42	M	2	4	144	98	62	169	21.7	84	116	0.72	37.5	198	226	0.31	172	148	NO EVIDENCE	0
79	MAMUDA	50	F	2	7	134	82	56	149	25.22	88	102	0.86	34	247	289	0.28	198	162	NO EVIDENCE	0
80	VIJAYAN	49	M	2	12	140	80	68	160	26.5	99	108	0.91	38	226	299	0.38	244	143	MILD NPDR	1
81	RAJENDRAN	52	M	2	7	140	80	54	164	20.07	76	94	0.8	34	203	288	0.24	194	137	NO EVIDENCE	0
82	VASUMATHI	29	F	1	7	128	72	64	162	24.38	82	96	0.85	34	161	193	0.32	169	142	MILD NPDR	1
83	JAYALAKSHMI	44	F	2	7	138	82	62	158	24.83	90	108	0.83	34	192	276	0.17	186	134	NO EVIDENCE	0
84	SATHYABAMA	51	F	2	12	154	94	54	150	24	87	92	0.94	32	143	175	0.19	222	158	NO EVIDENCE	0
85	KOTAGIRI	52	M	2	10	148	88	98	174	32.36	114	122	0.93	41	185	202	0.48	282	214	MODERATE NPDR	2
86	VENKATESAN	58	M	2	8	142	88	68	161	26.23	86	98	0.87	46	243	287	0.22	248	179	NO EVIDENCE	0
87	SANKARAN	52	M	2	7	128	76	62	168	21.96	86	102	0.8	35	146	285	0.32	232	155	NO EVIDENCE	0
88	ANANDA NAIDU	56	M	2	5	148	90	56	164	20.82	83	104	0.79	36	116	211	0.24	254	139	NO EVIDENCE	0
89	MAHABUBEE	60	F	2	8	136	82	56	149	25.22	89	96	0.92	30	89	123	0.12	241	162	NO EVIDENCE	0
90	SABEERA BEGAUM	56	F	2	4	136	74	62	152	26.83	76	95	0.8	32	144	168	0.21	214	158	NO EVIDENCE	0
91	RENGANATHAN	54	M	2	10	134	72	62	160	24.21	96	100	0.96	35	162	198	0.28	244	159	MILD NPDR	1
92	THIRUPURAM	54	F	2	12	116	74	45	146	21.11	70	99	0.7	32	211	257	0.19	245	139	NO EVIDENCE	0
93	KUPPAMMAL	62	F	2	6	138	84	79	164	29.37	104	108	0.96	37	252	289	0.64	218	143	MILD NPDR	1
94	MOHAMMED	39	M	2	10	154	92	72	160	28.12	98	106	0.92	34	218	253	0.32	154	168	NO EVIDENCE	0
95	PRABAVATHI	50	F	2	12	126	76	88	160	34.375	90	103	0.87	31	222	286	0.54	198	138	NO EVIDENCE	0
96	VARADHAN	55	M	2	11	128	80	92	168	32.59	102	112	0.91	38	208	316	0.41	292	188	MODERATE NPDR	2
97	ZAHITHA	52	F	2	7	142	94	88	148	40.17	110	124	0.88	42	278	332	0.37	266	182	MILD NPDR	1
98	ELLAMMAL	54	F	2	12	120	88	65	154	27.4	99	104	0.95	36	216	248	0.32	259	169	MODERATE NPDR	2
99	JEYARAMAN	60	M	2	7	142	80	70	168	24.8	94	102	0.92	34	264	392	0.16	262	164	MILD NPDR	1
100	BALU	54	M	2	8	138	78	62	166	22.49	76	88	0.86	33	214	258	0.06	217	189	NIL	0